Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

AVMA Convention—Philadelphia, August 18-21, 1958

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Correspondence

May 19, 1958

Dear Editors:

It is not my custom to write letters of this sort, but I was strongly moved to do so after reading Brewer's article on delayed action quinacrine on tapeworms in dogs (*J.A.V.M.A.*, April 15, 1957: 340-342). Let me hasten to add that I have published, that I know editing is tough work, and that I bear no malice toward editors.

After reading the article, there seemed to be some elements missing. Further digging revealed that:

1) Table 1.—No efficacy by species is indicated; hence, what *does* the table mean?

The number of dogs held to 45-60 days are really very few (5).

2) Table 2.—The original 6 dogs are reduced to 3 by: (*a*) virtue of the short-time interval from dose to sacrifice which interval is determined by the species involved; especially Dipylidium, clearly indicated by Enzie, *et al.* 1957 b, whom Brewer has read. The remaining dogs are hardly sufficient data.

b) No species of tapeworms is indicated, therefore the results in the table are essentially unknown to me.

3) Results (page 341) of 103 dogs tested (80 to test its side effects)—I was very discouraged to find no data offered on the dosage in the 80 dogs.

4) Discussion (page 342)—I am not aware that rabbit feces are at all infective for dogs with regard to either Taenia or Dipylidium.

Having had some experience with both quinacrine and Keybrin, I feel that I have a better reason to ask questions than a casual reader. Incidentally, my results on efficacy and side effects with Keybrin are quite different from Brewer's results. Even if I had had no experience, I believe some basic principles of presentation have been violated. Since I have had the same sort of thing called to my attention by members of your editorial board, I am now very interested in learning how the article under discussion saw the light of day. I, like yourself, am interested in J.A.V.M.A. papers being of excellent character.

Very cordially yours,

s/FRANK EHRENFORD, Ph.D.,
Parasitologist,
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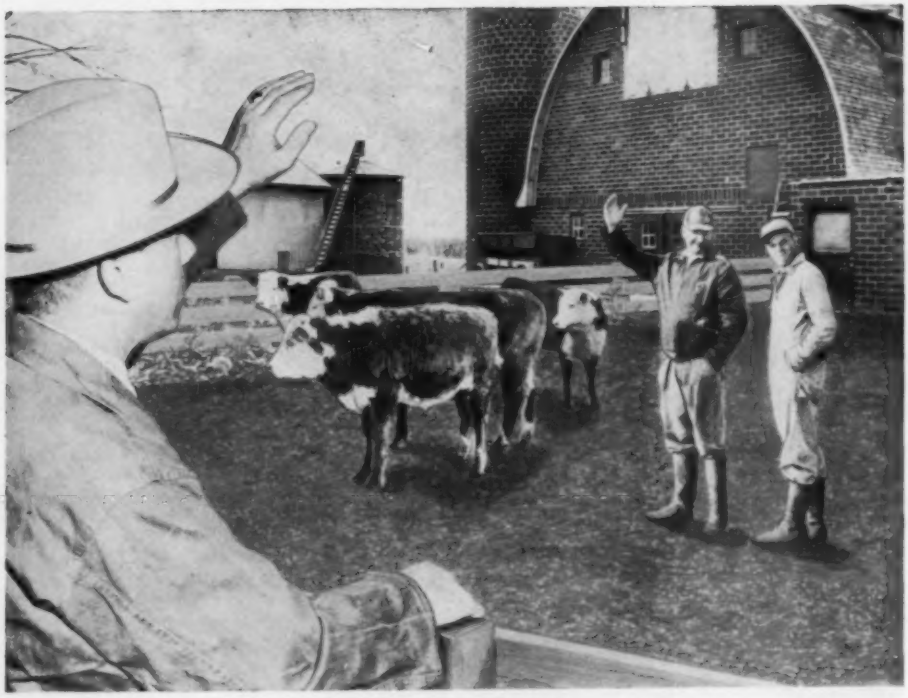

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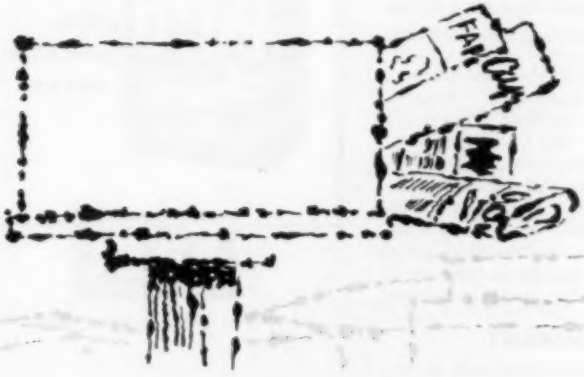

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Washington News

Legislative.—U.S.D.A. appropriations for fiscal 1959 (P.L. 85-459) include for ARS: all research, $59,044,890 (increase $1,250,000); Plant Animal Disease and Pest Control, $41,132,000 ($21,050,000 increase includes $20,000,000 for brucellosis program); Meat Inspection, $17,326,000 (increase $500,000). Agricultural Marketing Service, Poultry Inspection, $7,000,000. Conferees of the Senate and House agreed that the new mandatory poultry inspection may be located under A.M.S. (see JOURNAL, April 15 and May 15, 1958, adv. p. 8).

Federal Employees Salary Increase Act of 1958 became P.L. 85-462 on June 20, 1958, providing a 10 per cent increase retroactive to Jan. 1, 1958.

Each civil service employee entitled to receive an annuity, based on service which terminated prior to Oct. 1, 1956, will receive an increase (effective Aug. 1, 1958) of 10 per cent but not to exceed $500 per annum. Among the beneficiaries of the legislation, which had active support of AVMA, are many retired veterinarians.

Senate Committee on Government Operations by adversely reporting Senate Resolution 297, June 17, 1958, assures the merger of O.D.M. and F.C.D.A. effective July 1, 1958. The President, on June 25, announced the appointment of Honorable Leo A. Hoegh, now Civil Defense Administrator, to head the new office.

House Ways and Means Committee conducted hearings June 16 to 27 on some 400 bills pending on various aspects of the Social Security Act. In a press release, the chairman, Honorable Wilbur Mills, emphasized that it might not be possible to act on all proposals, but the testimony presented would be available for study and as a possible basis for action in the next session.

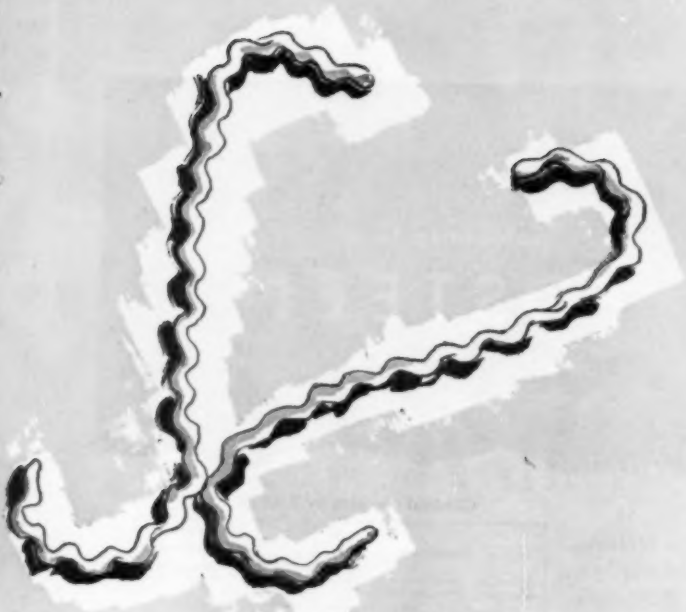
Senate Committee on Agriculture expects to schedule hearings on H.R. 12126 (see JOURNAL, June 15, 1958, adv. p. 14). Purpose of the bill, amending Act of June 17, 1930, is to extend to wild animals, which may be carriers of foot-and-mouth disease or rinderpest, the same prohibition against entry into the United States from infected countries as now applies to domestic animals.

New Bills—H.R. 12875 and H.R. 12876, identical bills, introduced by Rep. John Bell Williams (D., Miss.) and Kenneth A. Roberts (D., Ala.) respectively, would amend Title VII of the Public Health Service Act (relating to health research facilities) and for other purposes. Bills would add five more years and an authorized $150 million to the medical research construction program, and includes an amendment so that grant money could be used to build medical and dental school facilities, latter at discretion of the Surgeon General and his Advisory Council. H.R. 1289.

★ ★ ★ ★

Miscellaneous.—Military Credit—AVMA Convention: approved for eligible veterinary reserve officers (see JOURNAL, June 15, 1958, adv. p. 14).

The U.S. veterinary delegation to the Soviet Union is one of six agricultural groups U.S.D.A. is sending to the U.S.S.R. this summer and fall, with the Soviet Union sending the same number of delegates to the United States. The delegation exchange plan is in accordance with an agreement between the United States and the Soviet Union for a general program of exchange in cultural, technical, and educational fields. Members of each U.S. group will come from U.S.D.A. and Land-Grant institutions. The veterinary science delegation, departing on or about July 15, 1958, will be: Drs. W. A. Hagan, Cornell University, and Rue Jensen, Colorado State University; and from ARS: Drs. A. H. Frank, F. D. Enzie, J. J. Callie and C. D. Van Houweling. The objective of this group is to obtain information on basic and applied research on diseases and parasites of animals.



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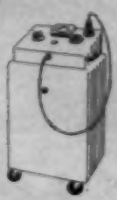

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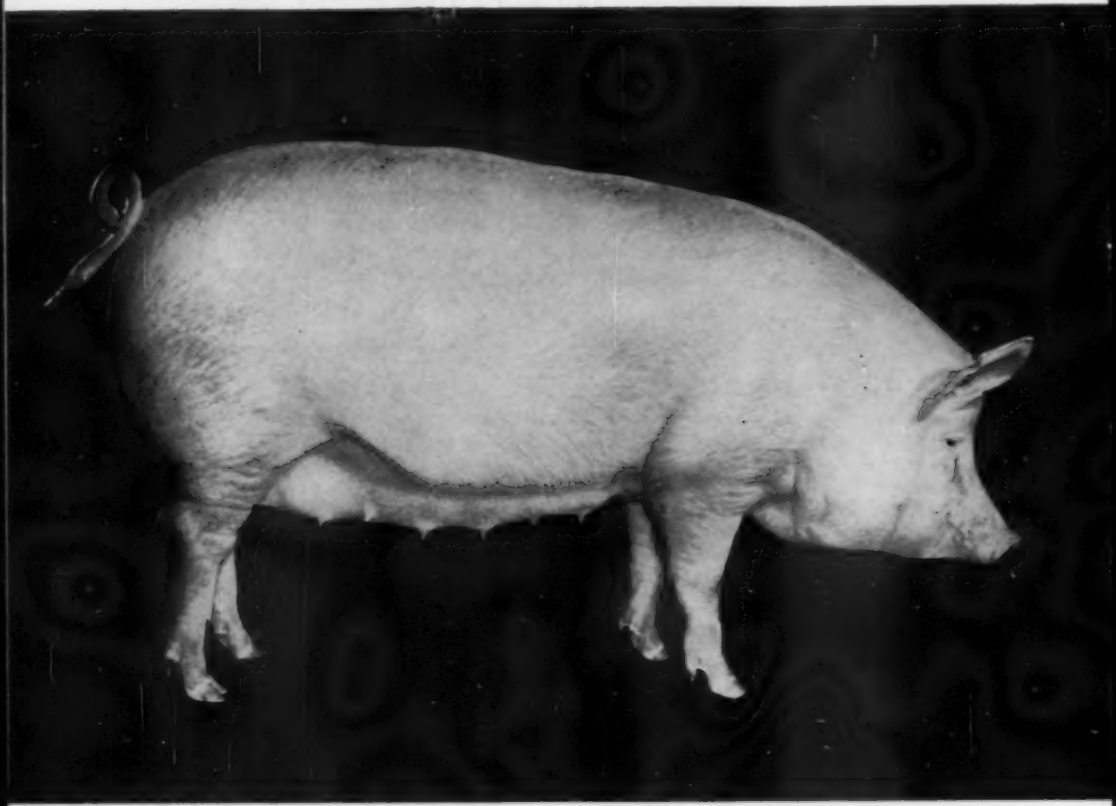

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The Efficacy of the Brucellosis Ring Test in Certifying Areas

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THIS PAPER is presented to provide additional information concerning the ability of a program of semiannual brucellosis milk and cream ring tests to detect dairy herds infected with Brucella. Questions are continually arising regarding the reliability of using negative ring test results in calculating the percentage of reactor herds and cattle for the purpose of certifying or recertifying counties as modified-certified brucellosis-free.

Another question concerns the effectiveness of a program of semiannual ring tests combined with "partial" blood tests every three years for the purpose of recertifying areas. Partial blood tests are tests done on all infected and suspicious herds scheduled for retests and on herds that do not have a history of negative ring tests during the previous year.

It has been shown previously that the brucellosis ring test is effective in locating infected dairy herds. Data relating to its effectiveness were tabulated and published in 1951 for nine Minnesota counties.[1] These tabulations showed that three or four semiannual ring tests resulted in the location of approximately 85 per cent of infected dairy herds containing approximately 95 per cent of the reactor dairy cattle.

The results of a survey conducted in Isanti county, in 1954, substantiated the above findings.[4] The initial area blood test in Isanti county, in the fall of 1953, was preceded by four ring tests at intervals of about six months. The area blood test involved 1,125 herds and 25,497 cattle, with the disclosure of 244 (23%) reactor herds (including 5 beef herds) and 673 (2.6%) reactor cattle. The four ring tests preceding the initial blood test failed to disclose 21 (14%) of the total reactor dairy herds containing 27 (4%) of the total reactor dairy animals.

One of the most important points regarding the value of semiannual ring tests was illustrated again by the results of the first ring test six and a half months after the countywide blood test in Isanti county. This ring test resulted in the disclosure of 19 new reactor herds (negative to the blood test six and one half months previously) containing a total of 43 reactor animals.

The early disclosure of such new centers of infection by a program of semiannual ring tests is important from the standpoint of the control and eradication of bovine brucellosis.

METHOD

To obtain the data presented here, the results of countywide blood tests for 14 predominantly dairy counties were tabulated to determine the unknown infection that would have remained if partial blood tests had been made instead of complete tests. The 14 counties included six northern Minnesota counties that have been modified-certified brucellosis-free for many years, thus representing areas with low levels of infection and of spread of infection. Their last complete blood tests had been about six years previously.

The other eight counties were not certified and represented areas with significant-

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ly higher levels of infection and of spread. This was their third complete area blood test, the two previous tests having preceded this one at intervals of approximately 18 months.

The data were obtained by checking the lists of infected herds, found on the countywide blood tests, against (1) the herd ring tests for the preceding year, (2) the first ring test following the area blood test, and (3) the previous herd blood tests, to determine whether such herds would have been included in a partial test.

RESULTS

In tabulating the countywide blood tests, in six certified counties (table 1), herds listed under the heading "Reactors in ring test-negative herds" represent the number of reactor herds and cattle that would not have been disclosed by a "partial" blood test in these counties, *i.e.*, by tests of only the beef herds, infected and suspicious herds, and those herds that did not have a history of negative ring tests the previous year.

The percentages in the column "Serum plate test titers" refer to the total number of reactor animals in ring test-negative herds. All other percentages in the table were calculated on the basis of the total number of herds and cattle tested.

The results of the countywide blood test in the eight noncertified counties are similarly shown (table 2).

DISCUSSION

The 1.88 per cent reactor herds and 0.20 per cent reactor cattle disclosed in the six certified predominantly dairy counties (table 1) indicate that a program of semiannual ring tests was effective in holding the infection at a level which is significantly below that permitted for recertification.

Partial blood tests in these counties, *i.e.*, omission of tests on herds with negative ring test histories, would have left undisclosed 1.23 per cent reactor herds and 0.09 per cent reactor animals in the counties. The latter percentage (0.09%) is nearly one half of the reactor animals (0.20%) found in these six counties. However, there are several indications that an appreciable percentage of these may not have been infected animals. These indications are as follows:

1) The pattern of distribution of titers for the 90 reactors in the ring test-negative herds (61% at + 100 and 11% at + 200 or higher) was abnormal for reactor animals, in general, in more heavily infected areas. The distribution of serum titers for 204 recently branded reactors, selected at random at the South St. Paul stockyards in the summer of 1956, and for 288 reactor animals found in obviously infected herds (2 or more reactors per herd), selected at random from recent countywide blood tests, was 13 per cent at + 100 and 74 per cent at + 200 or higher.

The pattern of titers for the reactors

TABLE 1—Results of Countywide Blood Tests in Six Minnesota Certified Counties*

	Countywide blood test results				Reactors in ring test-negative herds				
	Total tests		Total reactors		Reactor herds and cattle		Serum plate test titers		
County	No. herds tested	No. cattle tested	No. reactor herds	No. reactor cattle	No. reactor herds	No. reactor cattle	I + + No.	I ** + + No.	+ + + No.
Aitkin	1,363	21,265	30	34	16	16	14	2	0
Beltrami	1,362	18,959	12	27	5	5	1	4	0
Clearwater	1,086	16,984	34	49	26	29	19	7	3
Hubbard	765	11,452	9	11	6	6	3	1	2
Koochiching	677	8,023	13	32	4	5	3	2	0
Roseau	1,631	22,146	41	43	28	29	15	9	5
Total	6,882	98,829	129	196	85†	90	55	25	10
Per cent of total herds or cattle			1.88%	0.20%	1.23%	0.09%	61%‡	28%	11%
Single reactor herds					81	81	51	21	9
Per cent of total herds or cattle					1.18%	0.08%	63%	26%	11%

*Interval between countywide tests was approximately six years; **dilutions of 1:50, 1:100, and 1:200; − = no agglutination, I = incomplete agglutination, and + = complete agglutination; †includes three herds with 2 reactors each and one herd with 3 reactors; ‡percentages of the total number of reactor animals in ring test-negative herds.

found in the ring test-negative herds in the eight noncertified counties (table 2) was 21 per cent at + 100 and 64 per cent at + 200 or higher. The only obvious reason for the different pattern of titers in the six certified counties was that a high percentage of the reactions must have been nonspecific reactions.

2) Limited studies on the use of the acidified plate antigen (APA),[2,3] to distinguish between specific and nonspecific agglutination reactions, indicate that probably not more than 30 per cent of the 81 single reactor animals missed by the ring tests in the six certified counties were infected with Brucella.[4] If this is true, the probable number of infected animals missed by the ring tests might be considered as 28 animals or 0.03 per cent of the cattle in the six counties. This percentage is comparable to the value of 0.04 per cent for reactor animals missed by the ring tests in the eight noncertified counties (table 2).

The high percentage (64%) of + 200 or higher reactions obtained on the reactor animals missed by the ring tests in the noncertified counties indicates that a high percentage of the animals were infected. The results obtained with APA tests on reactor animals of this type in noncertified areas also indicate a high percentage of infection.

In this summary, predominantly beef or mixed herds (beef and dairy) were considered as dairy herds if ring tests were obtained on them. Although ring tests are of considerable value in detecting infection in such herds, they can not be as reliable as for strictly dairy herds, since they represent tests on milk from only part of the herd. In a plan of control and eradication of brucellosis in which semiannual ring tests are a part, provisions should be made for blood tests, at suitable intervals, of the mixed herds as well as beef herds, even though the former may have histories of negative ring tests.

The importance of including mixed herds in a partial test, irrespective of their ring test histories, is illustrated by the results of a recent countywide blood test in a county that contains an appreciable number of mixed herds. This test involved 2,318 herds and 38,033 cattle and disclosed 15 (0.65%) reactor herds and 37 (0.10%) reactor animals.

Seven of the 15 reactor herds, containing 21 of the 37 reactor cattle, were negative to previous ring tests and would have been missed by a partial blood test which omitted tests on herds with negative ring test histories. However, six of the seven herds were listed as Hereford, Shorthorn, or mixed, and only one was listed as a dairy herd. The latter herd contained only 1 re-

TABLE 2—Results of Countywide Blood Tests in Eight Minnesota Noncertified Counties*

County	Countywide blood test results: Total tests: No. herds tested	Countywide blood test results: Total tests: No. cattle tested	Countywide blood test results: Total reactors: No. reactor herds	Countywide blood test results: Total reactors: No. reactor cattle	Reactors in ring test-negative herds: Reactor herds and cattle: No. reactor herds	Reactors in ring test-negative herds: Reactor herds and cattle: No. reactor cattle	Reactors in ring test-negative herds: Serum plate test titers: 1 + + No.	Reactors in ring test-negative herds: Serum plate test titers: 1 ** + + No.	Reactors in ring test-negative herds: Serum plate test titers: + + No.
Carver	1,622	46,926	74	226	18	20	6	2	12
Dakota	1,233	30,794	57	121	6	9	1	3	5
Hennepin	1,284	33,461	75	156	11	15	4	1	10
Isanti	1,102	21,779	44	118	6	7	4	2	1
Kanabec	1,094	25,700	50	136	11	14	5	1	8
Stearns	3,908	104,736	193	653	28	42	4	6	32
Washington	1,025	25,829	41	90	6	6	2	2	2
Wright	2,743	68,018	65	243	17	30	4	5	21
Total	14,011	357,243	599	1,843	103†	143	30	22	91
Per cent of total herds or cattle			4.27%	0.51%	0.73%	0.04%	21%‡	15%	64%
Single reactor herds					84	84	17	15	52
Per cent of total herds or cattle					0.60%	0.02%	20%	18%	62%

*Interval between countywide tests was approximately 18 months; **dilutions of 1:50, 1:100, and 1:200; − = no agglutination, I = incomplete agglutination, and + = complete agglutination; †includes ten herds with 2 reactors each, four herds with 3 reactors each, two herds with 4 reactors each, two herds with 5 reactors each, and one herd with 9 reactors; ‡percentages of the total number of reactor animals in ring test-negative herds.

actor with a + 100 serum titer and a negative reaction to the APA test.

Some of the reactor herds with negative ring test histories, in the 14 counties, may represent new infection which developed in the interval between the ring test and the area blood test—an average interval of about two and one half months for the 14 counties. The elimination of the reactors disclosed by the area blood test may have been responsible, in some instances, for negative ring tests on such herds on the first area ring test that followed the area blood test.

There is little probability of obtaining a negative ring test on a herd containing 2 or more infected animals in milk production. A study of the ring test titers on individual milk samples from 303 reactor cows showed that 80 per cent of such samples could be diluted 1:25 with mixed negative milk and still obtain a 2+, or stronger, ring test reaction.[4]

On this basis, the probability of obtaining a positive ring test reaction on mixed milk from 25 cows, when 2 of them are reactors, is 96 per cent, and it is 99 per cent when milk of 3 reactors is included. It would seem that some of the herds, containing 2 or more reactors, which were negative to previous ring tests, might have been suspicious to a ring test if such a test had been made at the same time as the blood test.

The data presented in this report, along with those reported earlier for a similar study,[1] represent the data obtained from 19 countywide blood tests in predominantly dairy counties, including 26,247 herd tests and 580,486 animal blood tests. The data are such that there should be little doubt that dairy herds which maintain a negative ring test history under a program of semiannual ring tests may, with considerable safety, be considered as negative for purposes of certification or recertification of a county as modified-certified brucellosis-free. This is true particularly under conditions where there are provisions for blood tests, at suitable intervals, of all beef and mixed herds and all dairy herds not in the ring testing program.

SUMMARY

1) The results of countywide blood tests in six counties certified as modified-certified brucellosis-free and in eight noncertified counties were checked against the ring test histories of the herds. The number of reactor animals found in herds with negative ring test histories in the six certified counties represented 0.09 per cent of the total number of animals tested in those counties. The corresponding value for the eight noncertified counties was 0.04 per cent.

2) The data indicate that dairy herds which maintain negative ring test histories under a program of semiannual ring tests may, with considerable safety, be considered as negative for purposes of certification or recertification of a county as modified-certified brucellosis-free. This is particularly true when all beef and mixed herds and all dairy herds not in the ring testing program are blood tested at suitable intervals.

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[1]Driver, F. C., and Roepke, M. H.: Results of Countywide Blood Tests Following Two to Four Countywide Ring Tests for Brucellosis. Proc. Book, AVMA (1951): 148-153.

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Shoeing Oxen.—Of 15.6 million cattle in eastern and western Germany, in 1957, about 1.7 million cows, bulls, and oxen were used as draft animals and cloven-hoof shoeing is common. Durable spring-iron shoes, one for each digit, are shaped to cover the sole with a strap bent so as to come up on the axial surface and bend over the dorsal surface of the hoof. Each is held in place with nails placed similarly to those used in shoeing horses.—*K. Drawer in Berl. u. Münch. tierärztl. Wchnschr. (April, 1958): 124.*

Fowl Cholera in Ducklings.—Of 320 ducklings, about 3 weeks old, 287 died of pasteurellosis. Adult ducks, geese, and fowl on the same farm remained healthy. The other 34 ducklings were given *Pasteurella aviseptica* serum and survived.—*Vet. Bull. (April, 1958): Item 984.*

The Blood Picture in the Race Horse. I. The Normal Erythrocyte and Hemoglobin Status—A Dynamic Concept

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WHILE STUDYING anemia in horses, I noticed that, on exertion, a horse might show signs suggestive of anemia in spite of the fact that its hemoglobin level and erythrocyte count were no lower than that of the average horse. To determine the level at which hemoglobin and erythrocyte counts could be regarded as indicative of anemia, the normal levels must first be known. The figures used for this (table 1) were taken as representative of those in the literature.

Race horses, as distinct from foals, brood mares, and yearlings, gave hemoglobin levels of 15.4[6] and 12.7,[11] respectively. This variation in standards made the definition of the margin between anemia and normality even more difficult, Trum's apparently normal horses being anemic by Macleod and Ponder's standards.

Frequency of response to treatment has lead me to regard anemia as a fairly common condition in the race horse—a condition which is most significant when maximum muscular activity is required. It is important, therefore, to define accurately the hemoglobin level and erythrocyte count in the normal race horse.

During the seven years ending in 1954, I examined the blood of 264 Thoroughbred and Standardbred horses; these included 40 horses which were under constant supervision and were subjected to weekly examinations. Tests were also made of the horses after excitement, after exercise of various grades of severity, and at regular intervals until findings returned to normal; also after a blood transfusion, hemorrhage, and after administration of adrenaline or of drugs which were thought likely to affect blood levels.

The purpose of this paper is to define the hemoglobin levels and erythrocyte counts in the normal race horse.

TECHNIQUE

For each test, 5 cc. of blood was withdrawn from the jugular vein of the horse into bottles containing 1.3 mg. of ammonium oxalate and 0.7 mg. of potassium oxalate. Examinations were made almost immediately after collection. Hemoglobin estimations were made with the electrohemometer. Erythrocyte counts were not found sufficiently accurate for the investigations which followed; many identical estimations were often made on the one sample. The highest and lowest figures never differed by more than 2 per cent for packed cell volume (PCV) or more than 4 per cent for hemoglobin, but differed up to 15 per cent in erythrocyte counts.

That other workers have had similar irregularities in erythrocyte counts is apparent. Hansen *et al.*[2] repeated erythrocyte counts until they had two counts within 500,000 of each other, and then averaged these two counts.

A comparison of the indexes of mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) given in the literature shows that MCHC derived from hemoglobin and PCV is remarkably constant, whereas those derived from erythrocyte counts show great variation.

The strong tendency to pseudoagglutination in the horse may be the cause of the difficulty in obtaining accurate erythrocyte counts and, unless the blood is mixed thoroughly before sampling, inaccuracies in other estimations also. Hemoglobin levels and PCV were used in most investigations and are designated "erythrocyte concentrations."

DETERMINATIONS UNDER VARIOUS CONDITIONS

Resting Levels.—My figure of 11.4 for the hemoglobin level in 184 horses, 3 years old and over, in training (table 2), is much lower than that of Macleod and Ponder[5] of 15.4 for 4-year-olds and over. However, figures as high as those of the other workers were recorded when the horses were moderately excited. These determinations were not included in arriving at the figure of 11.4, as the horses were not regarded as being in the true resting state.

The 184 estimations were made on horses where we could be certain that they were in true basal state before blood samples were taken.

Twenty-four hours after exercise, erythrocyte concentrations were still well above normal (table 5) so samples were taken on the evening of the day of rest.

I have used only clinically normal horses, but can not exclude the possibility that some subclinically anemic horses may have

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The author thanks Drs. B. Howie, Otago Medical School, and N. G. Prentice, Southland Hospital, for their assistance.

TABLE 1—Normal Hemoglobin and Erythrocyte Levels of Thoroughbred Horses (as reported in the literature)

Reference	No. of horses	Hb.* (Gm./100 cc.)	Eryth. (million/cc.)	PCV (%)	MCV (%)	MCH (Gm./-100 cc.)	MCHC (Gm./-100 cc.)
Macleod and Ponder (1946)[8]							
Foals	8	13.9	12.9	.....	.....	10.7	.....
Yearlings	12	14.1	12.5	.....	.....	11.4	.....
2- and 3-year olds	12	14.1	10.8	.....	.....	13.1	.....
Older	8	15.4	11.6	.....	43.2	13.3	32.7
Hansen et al. (1950)[2]							
Stallions	36	14.7	10.7	46.9	43.2	.....	31.2
Mares in foal	65	14.9	10.5	45.3	42.9	.....	32.9
Barren mares	70	13.9	10.0	42.8	43.2	.....	32.2
Trum (1952)[10]							
Foals	28	13.4	10.8	.....	.....	.....	.....
Yearlings	53	11.2	8.1	.....	.....	.....	.....
Older	.....	12.7	9.3	.....	.....	.....	.....

*Hb = hemoglobin; eryth. = erythrocytes: PCV = packed cell volume; MCV = mean corpuscular volume; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration.

been used. A subgroup of the best performing 30 horses gave the following figures: hemoglobin 12.0, PCV 35, MCHC 34.0, compared with the over-all average of 11.4, 36, and 31.7. The figures are almost identical, except that the selected group shows higher hemoglobin saturation of erythrocytes, which seems to be characteristic of the best horses.

After Excitement.—To examine the effects of excitement, horses were selected from stables visited only occasionally so as to simulate conditions under which previous workers obtained their figures. When a blood sample was taken, the needle was introduced without any special precautions to minimize excitement. The needle was left *in situ* and a stopcock was attached and filled with heparinized saline solution, or a tightly fitting stilet was introduced.

Further samples were taken at intervals until three consecutive estimations showed a difference of not more than 4 per cent. This was believed to be the basal level. The animal was then twitched and spoken to roughly, or forcibly restrained, and a further sample taken. After an interval, 2 cc. of adrenaline in a dilution of 1:1,000 was injected intravenously and blood samples were again collected at intervals.

While there was some variation attributable to temperament, the general trend was the same. The horse, Chief Light, was a typical example (table 3).

It was found that even the slight excitement caused by venepuncture by a stranger could cause an increase in erythrocyte concentration of 10 to 15 per cent and, with some conditions and temperaments, much more. It is apparent that samples taken when horses are restrained can be misleading. It might be argued that a true basal sample is not necessary for routine work. However, blood samples can be interpreted only by comparing them with a standard, and it is obvious that standard conditions must be observed when levels fluctuate so readily.

During excitement, an increase of 10 to 20 per cent in erythrocyte concentration was reported in grade horses[7] and of 28 and 15 per cent, respectively, in a Thoroughbred barren mare and weanling.

In the group "3-year-olds and over, not in training," horses on pasture were caught and blood samples taken and, although every effort was made to obtain basal conditions, it is probable that some degree of excitement persisted.

After Sedative Drugs.—Various seda-

TABLE 2—The Blood Picture of Race Horses in True Basal State

Designation	No. of horses	Hb. (Gm./100 cc.)	Eryth. (million/cc.)	PCV (%)	MCV (%)	MCH (Gm./100 cc.)	MCHC (Gm./-100 cc.)
Not in training							
2-year-olds	8	12.5	.....	.....	.....	.....	.....
3-year-olds and over	21	13.4	8.1	43.3	53.0	16.5	30.9
Brood mares	23	12.9	.....	.....	.....	.....	.....
In training							
2-year-olds	28	11.7	7.4	39.0	53.0	16.0	30.0
3-year-olds and over	184	11.4	6.7	36.0	53.0	17.0	31.7

TABLE 3—The Blood Picture of 1 Horse (Chief Light) After Venepuncture, Twitching, and Administration of Adrenaline

	Hb. (Gm./100 cc.)	PCV (%)	MCHC (Gm./100 cc.)		Hb. (Gm./100 cc.)	PCV (%)	MCHC (Gm./100 cc.)
Venepuncture	12.7	39.4	31.0	After twitching	14.9	.....	.....
After 5 min.	12.3	.....	.....	After adrenaline			
10 min.	11.8	.....	.....	3 min.	19.8	69.3	28.6
15 min.	11.4	.....	.....	10 min.	18.4	65.0	28.3
20 min.	11.3	.....	.....	30 min.	15.6	55.3	28.2
30 min.	11.3	35.9	31.8	60 min.	14.4	48.7	29.8

tives were used in an attempt to obtain true basal levels in excitable horses. Several drugs and drug combinations were tried and, of these, hydroxyzine hydrochloride (Atarex*) and benactyzine hydrochloride (Suavitil*) gave the most satisfactory results.

Results as low as, or lower than, those shown in table 2 were obtained when the animal was under light to moderate sedation.

In these excitable animals, a defense reaction followed handling and removal of blood, but under light sedation they soon returned to a basal state. However, the defense reaction appeared to make the animal confusedly conscious of its depressed state, and some incoordinated responses resulted in high erythrocyte concentrations. However, in all horses in which true sedation was obtained, levels approached those reported in table 2.

After Exercise.—No constant changes were observed[7] in the erythrocyte counts of horses exercised moderately. An average increase of 30 per cent in erythrocyte counts and PCV had been recorded[8] in horses after hunting, while increases of 20 per cent were recorded after trotting, with much greater increases occasionally. An increase of up to 37 per cent had been found[5] in draft horses after maximum labor, with normal or subnormal levels after 45 minutes.

A great variation was found among horses (table 4), and also in the same horse, at various ages and physical states. Only moderate exercise was required to give maximum response in the "high-strung" thoroughbred, though some of the more phlegmatic types required more strenuous exertion. Samples were taken immediately on completion of exercise to eliminate, as far as possible, the effects of sweating and diffusion of fluid out of capillaries. In some horses, maximum response was obtained with so little exertion that sweating could have had little effect.

Although searching for a correlation between performance and any single factor of anatomy or physiology is as fascinating and fruitless as selecting the winners by any other method, better staying performances were given by horses showing low basal erythrocyte concentration, with increase of 60 per cent or more under stress.

However, a surprisingly large number of horses failed to behave normally, showing either (1) high resting levels, (2) subnormal percentage of increase after stress, (3) low MCHC before and after stress, and other abnormalities. Although a full discussion of these conditions is outside the scope of a paper on normal levels and will be dealt with elsewhere, such abnormalities were definitely associated with poor performances.

Effect of Training.—In the normal race horse, regular training resulted in a marked increase in the erythrocyte count, which was found to be three times as high as in an animal not in training.[7] In this study, the erythrocyte concentration showed a slight drop with increasing fitness. In a group of 20 horses examined throughout their training, there was a drop from a pretraining level of 12.5 to 11.3 Gm. of Hb. per 100 cc. Occasionally, horses in training for long periods showed an increase in hemoglobin level, which frequently coincided with a falling-off in performance.

TABLE 4—Effect of Exercise on the Race Horse

Physical status	Hb. (Gm./100 cc.)	PCV (%)	MCHC (Gm./100 cc.)
Not in training			
Basal	13.4	43.3	30.9
After exercise	16.8	55.0	30.5
Trained horse			
Basal	11.4	36.0	31.7
After exercise	17.1	58.0	29.5

*Atarax is produced by Pfizer Laboratories, Brooklyn, N. Y.; Suavitil by Glaxo Laboratories, Ltd., Greenford, England.

TABLE 5—The Average Postexercise Curve (Hb. Levels) in Race Horses

Physical status	Hb. (Gm./100 cc.)	Physical status	Hb. (Gm./100 cc.)
Resting	11.4	After exercise completed	
After light preliminary exercise	16.1	15 min.	13.9
After 3 min. of exercise	17.1	20 min.	14.0
After exercise completed		45 min.	14.2
5 min.	15.5	2 hours	13.8
10 min.	14.6	24 hours	13.3
		48 hours	11.5

After Transfusion.—Up to 6 liters of compatible blood was given to normal horses. After a transitory rise, the hemoglobin levels usually returned almost to pretransfusion levels in 36 hours.

After Hemorrhage.—Blood samples were taken from several horses which were being "let down" after heavy training. Due to small numbers and wide variation in results from factors other than blood loss, the results would not stand up to critical statistical analysis. However, in several animals in which 1¼ to 1½ gal. of blood (20 to 25% of the total blood volume) was removed, the drop in basal erythrocyte concentration did not exceed 10 per cent in any horse at the end of 12 hours, most being 0 to 5 per cent (table 6).

TABLE 6—The Concentration of Erythrocytes Following Hemorrhage

Hemoglobin level (Gm./100 cc.)	Before bleeding	12 hr. after removal of 1 gal. of blood
Basal	11.8	11.4
After adrenaline	18.1	15.3

One race horse just out of training suffered severe accidental hemorrhage. Hemoglobin levels on this horse are shown (table 7). Five weeks after the accident, the MCHC was unusually high; such cases should be studied to determine the importance of this phenomenon. During the five weeks, a hematinic was given at high dosages.

TABLE 7—Hemoglobin Levels of a Horse After a Severe Hemorrhage

Status	Hb. (Gm./100 cc.)	PCV (%)	MCHC (Gm./100 cc.)
2 weeks before hemorrhage (slightly excited)	12.5	37.0	33.0
1 day after hemorrhage			
Basal	5.4	21.0	26.0
After adrenaline	7.5	28.0	27.0
5 weeks after hemorrhage			
Basal	15.4	31.0	50.0
After adrenaline	22.1	50.0	44.0

DISCUSSION

The hemoglobin level and erythrocyte count of the trained race horse are, I believe, in dynamic rather than static states, depending mainly on the condition (tone) of the sympathetic nervous system. Thus, a blood test reflects this condition at the moment of sampling, rather than the true erythrocyte and hemoglobin status of the animal. A sympathetic (quiescent) condition maintains the erythrocyte concentration at a level sufficient for the requirements of the moment, and increases of 60 per cent can occur within seconds.

The constant drop in erythrocyte concentration, in the resting state, with increased fitness is believed to be due to improved gaseous exchange and circulation in the lungs and tissues, so that lower concentrations suffice for normal resting functions. Low hemoglobin levels, then, could indicate that functions are most efficient and, provided the subject can mobilize sufficient erythrocytes for exertion, hemoglobin levels of 11.0 Gm. per 100 cc., or less, may not indicate anemia. (Incidentally, Olympic athletes at rest are reported to have hemoglobin levels significantly lower than normal.[10])

Conversely, high basal erythrocyte concentrations often have pathological significance, as may high heart and respiratory rates in resting animals. Increased basal erythrocyte concentrations were found after giving arsenic hematinics and were associated with subnormal increases after exercise, causing one to wonder how much of the "beneficial" stimulus to erythrocyte production is a result of the toxic effect on some other point along the chain of oxidative processes. Finally, horses with normal levels at rest may show hypochromic anemia on exertion, e.g., iron deficiency, vitamin B_{12} toxicity, and phenothiazine poisoning.[3]

The mechanism of the increase under stress will be dealt with in another paper. However, the conclusions reached have a

bearing on the adrenaline-control theory of erythrocyte concentration and will be mentioned briefly. Evidence will be produced suggesting that the increase is caused largely by mobilization of erythrocytes stored in the spleen and, to a slight degree, by lowering of plasma volume.

It has been shown[9] that in the horse, especially the race horse, the spleen has a much greater amount of contractile tissue than in any other animal. Contraction of the spleen is dependent on the condition of the sympathetic nervous system. Maximum contraction caused by strongest sympathetic stimulation is associated with maximum increase in erythrocyte concentration.

One day after severe hemorrhage in a horse, it was found that, although the horse was weak from oligemia, its hemoglobin increased 38 per cent after it was given adrenaline. This case was complicated by shock; however, it has frequently been observed that, although erythrocyte concentration may fall to a level at which it would appear to be to the animal's advantage to maintain all of these cells in circulation, up to 25 per cent are still held in reserve, once the hemorrhage has been controlled. This may be because mobilization is adrenaline-controlled, and only in cases of maximum adrenaline output, which usually occurs over a brief period, does complete mobilization occur. After 25 per cent blood loss, little drop in erythrocyte concentration at rest was observed. This indicates that, under these conditions, any work on the hematinic and hemolytic properties of drugs in the horse might be questioned.

I believe that the data of workers who fail to appreciate the large and extremely mobile erythrocyte reserves of the race horse must be of limited value, the wide variation in results being probably due to this.[5,10]

In the normal trained race horse, MCHC at rest is remarkably constant at 30 to 35 Gm. per 100 cc., and under stress at 27 to 30 Gm. per 100 cc. Incidentally, this drop in MCHC under stress is not due to systematic experimental error since, when critically checked by skilled technicians using instruments in which the error in each figure (Hb. and PVC) was less than 2 per cent, a drop of 10 per cent in MCHC still occurred in over 60 per cent of the horses. This drop was much less pronounced when the horse was not in the true basal state.

Discussion of the cause introduces findings outside the scope of this paper, including (1) mobilization of imperfectly hemoglobinized cells, both immature and effete, (2) mobilization of cells containing greater or lesser amounts of hemoglobin than those in the general circulation due to altered nutritional status, and (3) increases in the volume of the individual erythrocytes.

SUMMARY

Hemoglobin percentage, erythrocyte counts, packed cell volumes, and the indexes derived therefrom, are given for 264 race horses. Reasons for low resting levels are discussed.

The dynamic concept of the erythrocyte level is advanced. Attention is drawn to the fallacy of using the blood sample taken without regard to this as an indication of the blood picture, since low levels may indicate a high state of physical fitness rather than anemia, whereas a functional anemia may be accompanied by normal levels. Conversely, high resting levels are often pathological. The significance and mechanism of the increase under certain conditions is discussed. A normal state is defined as (1) basal levels as in table 2; (2) increase of at least 60 per cent under stress; (3) MCHC as in tables 2 and 4.

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Further Erythrocyte and Hemoglobin Studies in Thoroughbred Racing Horses

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SINCE OUR REPORT on the erythrocyte count and the hemoglobin concentration in 207 racing Thoroughbreds was published,[1] we have made a study of 359 additional racing Thoroughbreds, under training and racing conditions in southern California. The techniques for the various tests in this series were identical to those used in the initial report.

METHODS AND RESULTS

The places, time of study, and results were as follows: 208 horses studied at Hollywood Park, during the spring meeting, showed an average erythrocyte count of 6.805 million per cubic centimeter, an average hemoglobin concentration of 14.2 Gm. per 100 milliliters, and an average packed cell volume (PCV) of 43.6 mm.; 84 horses at Santa Anita, during the winter meeting, showed an average erythrocyte count of 7.212 million/cc., an average hemoglobin concentration of 14.1 Gm./100 ml., and an average PCV of 43.2 mm.; and 67 horses at Del Mar, during the summer meeting, had an average erythrocyte count of 6.643 million/cc., an average hemoglobin concentration of 14.2 Gm./100 ml., and an average PCV of 42.0 mm.

Apparently there is no appreciable or significant difference between these groups; the moderate changes in weather in southern California in the three areas seem to have little or no effect upon these physiological factors. However, this may not apply in areas of extreme cold or heat.

For all of the 359 horses studied, the average findings in whole blood were: erythrocyte count, 6.90 million/cc. (6.888); hemoglobin concentration, 14.0 Gm./100 ml.; PCV, 43.0 mm. (42.73). The extremes of the above were: erythrocyte count, 9.63 million/cc. and 4.50 million/cc.; hemoglobin concentration, 18.0 Gm./100 ml., and 9.7 Gm./100 ml.; and PCV, 58.0 mm. and 30.0 mm.

When the results of this work were correlated with that reported in the first paper, the average normal values for 566 Thoroughbreds under training and racing conditions were: erythrocytes, 6.84 million /cc.; hemoglobin, 13.86 Gm./100 ml., and PCV, 43.0 mm.

An excited horse is reported to have an elevated erythrocyte count.[2] In our present studies, this was found to be true in every instance where a Thoroughbred was disturbed, to any great extent, during the taking of the specimen for testing. Therefore, we decided to study these hematological changes just before and after racing to determine the extremes of hemoconcentration.

With the permission of the stewards, we obtained blood specimens from 4 "stake" horses two hours prior to their race and again within 15 minutes after the race. (table 1).

TABLE 1—Results of Blood Tests in 4 "Stake" Horses, Taken Two Hours Prior to Racing and Within 15 Minutes After Finishing

Horse No.	Eryth.* (million/cc.)		Hb.* (Gm./100 ml.)		PCV *(mm.)	
	Start	Finish	Start	Finish	Start	Finish
1	6.30	11.18	12.2	22.4	38.9	70.5
2	7.40	9.29	14.6	19.8	47.2	58.8
3	11.05	11.18	15.2	20.5	70.0	70.0
4	8.88	11.79	17.3	23.0	50.0	68.5

*Eryth.=red blood cells; Hb.=hemoglobin; PCV=packed cell volume.

Horse 1 entered the race with a blood picture below normal. It seemed that horse 3 must either be in a continual state of excitement or markedly dehydrated. It was later learned that the animal had been intentionally "drawn" for the race. Horses 2 and 4 appeared to be essentially normal prior to the race—they were well within normal limits.

The degree of hemoconcentration, how-

From the Brenon Laboratory, Inglewood, Calif.

ever, is clearly demonstrated in all animals at the termination of the race and would appear to indicate that a blood specimen would be valueless for study if it were obtained during workout or a race or during extremes of excitement.

Some of our further studies along this line seem to indicate that the cell count and hemoglobin concentration does not return to prerace levels until at least two hours after any race, workout, or moderate exercise.

We have stated[1] that clinically sound Thoroughbred horses that were not in training had higher erythrocyte and hemoglobin concentrations than horses that were under the daily stress of training and racing.

To substantiate this statement, we undertook a random blood study of 8 classified Thoroughbreds that had been on a rest farm for a minimum of 30 days and which had no clinical evidence of any type of dyscrasia.

These animals showed an average erythrocyte count of 7.66 million/cc.; an average hemoglobin concentration of 14.7 Gm./100 ml.; and an average PCV of 45.5 mm.

DISCUSSION

Presumably, an animal in training, with a controlled diet and feeding times and excellent general care, should be in superior physiological condition to an animal that is left to its own resources. However, this was not found to be true. Perhaps factors such as freedom of eating, type of feed, environment, and the unrestrained movement permitted on the farm may account for this difference. A study of these variables would seem to be indicated if horses are to produce their top performances.

In another study of 40 Thoroughbreds on the farm,[2] the average erythrocyte count was 10.35 million/cc.; the average hemoglobin concentration was 13.89 Gm./100 ml.; and the average PCV was 42.2 mm. However, the horses were of widely different ages and were not designated as racing Thoroughbreds at rest or under training. Random tests taken by us showed that horses that are rested on a farm periodically tend to show improved blood pictures.

Racing records of the horses evaluated in this study were not included because they were not considered to be of scientific importance, due to the fact that there are too many variable factors that come into play during the running of any one particular race. The study does, however, point out the normal values of one aspect of physiological well-being and, perhaps to some extent, stamina.

A routine study of the blood count of racing Thoroughbreds will assist both the veterinarian and the trainer to place, on the track, horses that are in most respects physiologically sound. It should be stressed that the interpretation of all findings should stay within the province of those persons qualified by training and practice to properly evaluate them under all types of variable conditions. Only then will the accurate blood picture prove of true value in the future of Thoroughbred racing.

It is our belief that the blood picture is one of the most valuable diagnostic tools with which to ascertain the potential physiological ability of an animal. A horse that is in excellent physical condition should be able to put forth its maximum effort. On the other hand, a horse may have an excellent blood picture but, due to even a minor dyscrasia of bone, joint, muscle, or temperament, may not be capable of maximum effort on its appointed racing day.

SUMMARY

1) In a study of 359 clinically normal Thoroughbred racing horses of both sexes, the average findings in whole blood were: erythrocytes, 6.90 million per cubic centimeter; hemoglobin concentration, 14.0 Gm. per 100 milliliters; and packed cell volume (PCV), 43.0 mm.

2) A combined evaluation of both studies, including a total of 566 Thoroughbred racing horses, appears to place the average normal erythrocyte count at 6.84 million/cc.; the average normal hemoglobin concentration at 13.86 Gm./100 ml., and the average normal PCV at 43.0 mm.

3) Any erythrocyte count below 6.50 million/cc.; any hemoglobin concentration below 13.0 Gm./100 ml., and any PCV below 40.0 mm. would seem to indicate that the horse is below its potential physiological peak.

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Dangerous Decline in Hog Cholera Vaccination

For a number of years prior to 1952, about 50 per cent of the pigs raised in the United States were vaccinated against hog cholera. The number has since declined to less than 40 per cent in 1957. While there is no evidence of an increased incidence of hog cholera, the greater number of susceptible herds would result in a more rapid spread and a more extensive epizootic if cholera should occur.

The hog cholera serum marketing agreement administered by the U.S.D.A. requires that commercial producers of serum have available, on May 1 each year, not less than 40 per cent of the serum they sold the previous year.—*U.S.D.A. (May 22, 1958).*

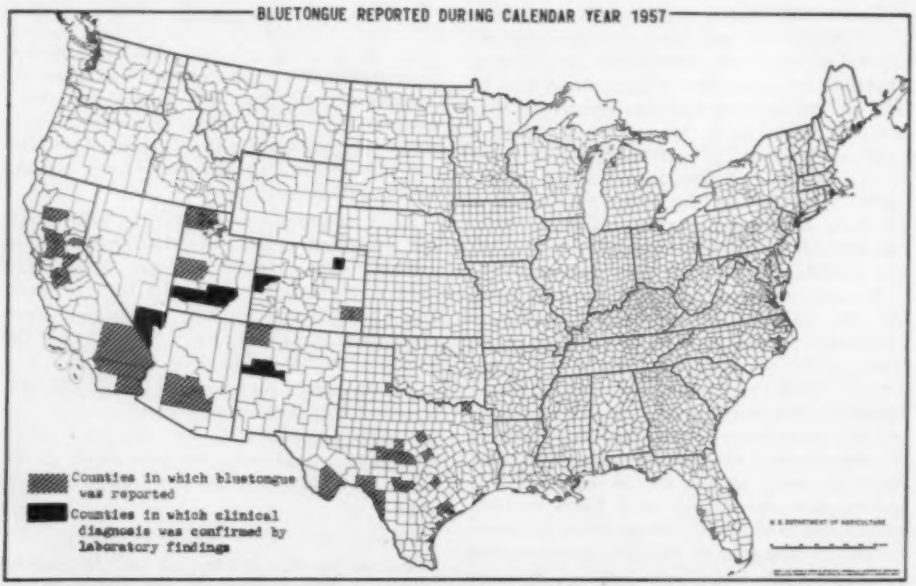
Hog Cholera Immunity in Pigs from Immunized Dams.—The immunity to hog cholera was of markedly shorter duration in pigs from sows which were immunized with lapinized virus at the time of breeding than from sows vaccinated with serum and virulent virus at breeding time.—*Vet. Bull. (April, 1958): Item 1063.*

Leptospirosis in Ohio Wildlife.—Since mid-1954, 932 blood samples from animals in Ohio have been tested for *Leptospira pomona*. Reactors were found in seven species but none in 150 rabbits, 37 muskrats, 9 cats, and in ten samples from four other species. Reactions ranging from 1:100 to 1:3,200 were found in 43 (19%) of 224 deer, 16 (23%) of 70 raccoons, 14 (25%) of 55 foxes, 6 (55%) of 11 skunks, and 1 each in 142 mice, 165 rats, and 43 opossums.

Bluetongue in the United States in 1957

Bluetongue, which seems to have occurred only in Texas prior to 1952, when it was recognized in California and Utah, was found in five states in 1953, ten states in 1954 and 1956, but only in the seven most southwestern states in 1957.

The morbidity in the affected flocks ranged from 0.1 to 43.0 per cent, being above 30.0 per cent in five states, and the


BLUETONGUE REPORTED DURING CALENDAR YEAR 1957
Counties in which bluetongue was reported
Counties in which clinical diagnosis was confirmed by laboratory findings

Map 1—Bluetongue was reported only in seven southwestern states in 1957. The following are states in which clinical diagnosis has been confirmed by laboratory findings: Arizona, California, Colorado, Kansas, Missouri, Nebraska, Nevada, New Mexico, Oklahoma, Oregon, Texas, Utah.

mortality among the affected sheep ranged from 0 to 33.0 per cent.—*U.S.D.A., March 19, 1958.*

Rabies in the United States in 1957

The 5,008 cases of rabies reported in animals in the United States, during 1957, was more than 800 fewer than in each of the previous two years. It was the lowest total number in 20 years and less than half the number reported in 1944 and again in 1946 (10,872). The trend in the change in the incidence for the species affected continued, with fewer canine and bovine cases and more rabies identified in wild animals. The 1,908 canine cases was approximately 600 less than in the previous two years, less than half of the 4,083 in 1954, and only 21 per cent of the maximum of 9,067 canine cases reported in 1944.

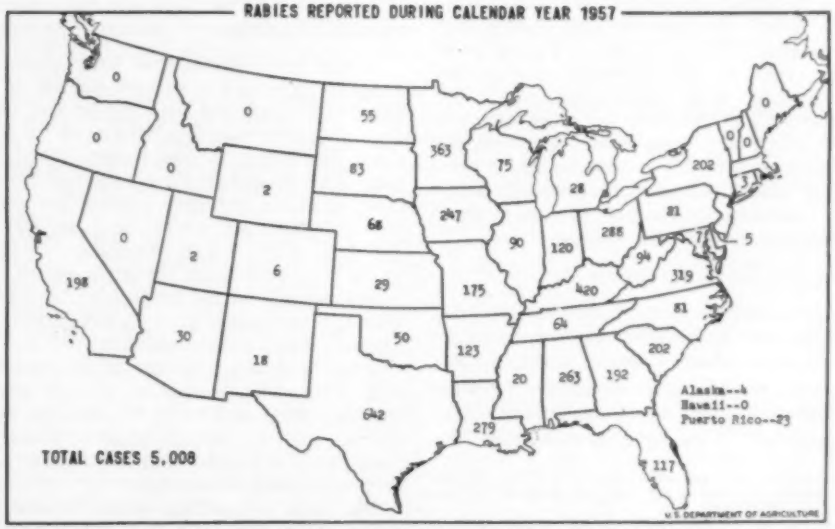
The 654 bovine cases was the smallest number since 1949 and considerably less than the 1,012 in 1953. The 1,989 wildlife cases has been exceeded only in 1956 and is more than twice the number ever reported prior to 1949. Since rabies in wildlife may often occur unobserved, these statistics are less accurate than for cases in domesticated animals. How much of the increase in number of cases reported is due to greater vigilance is unknown.

The 386 feline cases shows little fluctuation (207 to 538 cases) in this species in 15 years.

The percentage distribution by species in 1957 was: dogs, 38; foxes, 21; skunks, 15; cattle, 13; and cats, 8 per cent. There was a general downward trend in cases in all sections of the United States except the north central states.—*U.S.D.A., March 7, 1958.*

Aftosa in the Buenos Aires Zoo.—In 1942, 1948, and 1955, aftosa (foot-and-mouth disease) appeared in the Buenos Aires Zoo and affected cattle, buffaloes, deer, American bison, and grizzly bears. It was controlled by isolation of the animals and disinfection with 2 per cent sodium hydroxide.—*Vet. Bull. (April, 1958): Item 1038.*

Budgerigars and Poliomyelitis.—Several animals have been named as possible reservoirs for poliomyelitis virus but confirmation has always been lacking. In August, 1957, a budgerigar, which was recovering from paralysis of its legs, bit a boy on the lip. In seven days, the boy became ill and died of bulbar poliomyelitis in 13 days. The polio virus was isolated from the bird. Another bird in the same cage had died from a similar paralysis a few days earlier.—*J.Am.M.A. (May 3, 1958): 99.*



Map 1—There were 5,008 cases of rabies reported in animals in the United States in 1957 — the lowest number reported in 20 years.

Chemical Residues in Meat

R. K. SOMERS, D.V.M.

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The interest in chemicals in the diet has increased greatly in the last few years. Consumer attention, which has almost always been expressed as an aversion to chemicals in food, has been just as great as the interest of chemical manufacturers who have been diligently looking for new outlets for their products. In many cases, food manufacturers have also conducted extensive research aimed at producing improved or new food products by the use of chemicals.

The presence of chemical residues in food is naturally viewed with great concern by all food hygiene agencies and officials because consumers could receive considerable amounts of chemicals in their diets. Although a few chemicals used in foods are harmless, some are known to be harmful, and there are a great many chemicals about which little or nothing is known concerning acute or chronic toxic effects.

Our knowledge of long-used meat processing methods tells us that certain chemicals are commonly present in meat products such as cured meats and sausages. Research on the composition of meat has shown that even fresh meat sometimes contains added chemicals. We can, therefore, divide the subject into two parts: (*a*) chemicals added to meat during processing, such as salt, nitrates, nitrites, ascorbic acid, and phosphates; and (*b*) chemicals incorporated in meat through the biological process from the animal's environment, food, or treatment. Examples of the latter are insecticides, herbicides, hormones, antibiotics, therapeutic agents, and radioactive isotopes.

Development of Controls

The control of chemical residues in meat products has been effectively developed by the Federal Meat Inspection service for those chemicals which are added to meat during processing. Any meat packer whose products are federally inspected must furnish the Meat Inspection Division with proof of the safety of any new chemical proposed for use in the processing of meat. He must also prove that the chemical is necessary to produce a suitable product and does not result in adulteration or deception concerning the quality or character of the finished product.

An example of a chemical accepted for use in meat processing during the past few years is ascorbic acid, which aids in the production of the characteristic red color of cured products. However, the use of ascorbic acid on fresh meat has not been accepted because it has been found to result in a deceptive appearance of the meat.

The controls over chemicals which get into meat through the biological process are obviously more complex and are gradually being developed. There are indications that those who are discovering and developing such chemicals have not always given necessary attention to the effects that the chemicals might have on consumers of food derived from the treated animals or from animals consuming chemically treated feed. The consumers are not aware of the presence of these chemical residues because they do not usually cause any change in the appearance or character of the meat. However, many of these chemicals are known to be potent—as little as one part, or a few parts, per million in food can produce effects.

Possible Dangers

It has been shown that some chemicals have an affinity for certain tissues or organs when they get into the animal body. This characteristic has been used by clinicians for years in the treatment of disease. The chlorinated hydrocarbon insecticides such as DDT and lindane are examples of chemicals which have an affinity for fat. When these chemicals are used in excessive amounts for spraying or dipping livestock or for controlling insects on growing feedstuffs, they accumulate in the animal's fatty tissues quite rapidly and are eliminated slowly. The rate of accumulation and elimination varies with each chemical and may also be influenced by other factors such as species, state of nutrition, amount of exposure, and even some presently unknown conditions.

The data on toxicity which is available for certain chemicals has generally been developed through use of laboratory ani-

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Presented before the Public Health Section, Ninety-Fourth Annual Meeting, American Veterinary Medical Association, Cleveland, Ohio, Aug. 19-22, 1957.

mals and, for some chemicals, it is known that proof of the safety of using a chemical on food animals does not necessarily mean that the chemical is not harmful to those who eat meat from the animal.

It has been demonstrated that some chemicals exert a potentiation action when they are used together—in other words, the lethal effects of one chemical may be increased many times in the presence of another chemical. This action is especially noticeable in organic phosphorus insecticides which exert their lethal action by inhibition of cholinesterase—an essential enzyme which prevents the accumulation of excess amounts of acetylcholine in the body.

Radioactive isotopes are now getting much attention in the press. All living animals have some radioactive isotopes in their bodies—this was going on long before the "Atomic Age." All living things are constantly exchanging radioactive atoms, which act like nonradioactive atoms in the biological process. Strontium 90 has been headlined as the terrible poison from atomic fission which will cause untold damage to man and animals. Any theory given here about this could be argued endlessly but let us look at some of the presently known facts related to our food supply: Strontium 90 acts like calcium when taken into the body and is subsequently found in bones; consequently, since bones are not consumed by man, there would be little possibility of getting strontium 90 from this source.

Even if the ground where forage, vegetables, and grains are grown did become contaminated with this isotope, animals could be used as "strainers" to remove this source of danger from the food supply—naturally, this would result in some diet changes, but these would probably be of little significance compared to the other adjustments that would have to be made if there were a severe contamination of the world with strontium 90.

DISCUSSION AND CONCLUSIONS

It should be understood that the analytical methods necessary to detect chemical residues that may be deposited in meat animals through the biological process are in need of considerably more development. These methods require trained personnel and elaborate equipment for performing microchemical analysis. We have strongly recommended that research activities in the Department of Agriculture, and elsewhere, be emphasized in this field.

The Miller Amendment to the Food, Drug, and Cosmetic Act provides for the establishment of tolerances for residues of pesticide chemicals in raw agricultural commodities such as meat. The Federal Meat Inspection Division will not permit the mark of inspection to be applied to meat and meat products which are known to contain pesticide residues in excess of the tolerances established under the food and drug regulations. Products which do not come under the federal jurisdiction would be subject to state or local control.

The control of chemicals of the kind that are added to meat and meat products during their processing is adequate in federally inspected plants and in many state and locally inspected plants where regulations similar to the federal regulations are enforced.

Considerably more research is needed on those chemicals that get into meat through the biological system of the animal. Undoubtedly, some chemicals are necessary to economic production and, in such cases, research should be integrated with controlled use of the chemical until safe methods of use can be determined. The uncontrolled or indiscriminate use of chemicals on livestock or forage should be recognized as a dangerous practice which could result in serious effects on consumers of animal products.

Anthrax in Lions.—Anthrax was diagnosed at necropsy in 6 lions in a zoo in Germany. Hemorrhagic necrotic foci, typical of anthrax carbuncles, were found in the tonsils, intestinal mucosa, and lymph nodes in these areas, indicating infection through the alimentary tract with resulting septicemia.

Only the younger animals died but it is not certain that the older ones were fed meat of the same source. Deaths occurred from one to four days after the last possible time of infection. The 3 youngest lions had many intestinal parasites and 2 had subacute enteritis before the infection. Anthrax bacilli were found in the feces and urine of all 6 lions.—*H. Kronberger in Monatsh. f. Vet.-med. (April 15, 1958): 235.*

Ear Cropping by Triangulation

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THE FOLLOWING practical procedure for ear cropping has been used successfully on over a thousand dogs and has yielded a minimum of cropping problems.

The ideal age for ear cropping varies with the breed. In Boxers, for example, it is 8 to 12 weeks. At this age, a normal Boxer pup will begin to lift its ears laterally or cock them when its attention is attracted, an indication that it is developing the necessary muscle strength to hold the cropped ears erect. Dogs that are younger than 8 weeks or are malnourished are not considered good surgical risks and the owner should be advised to build up the animal before surgery is attempted. In general, in dogs that are over 3 months old, it is increasingly difficult to get the ears to stand following surgery.

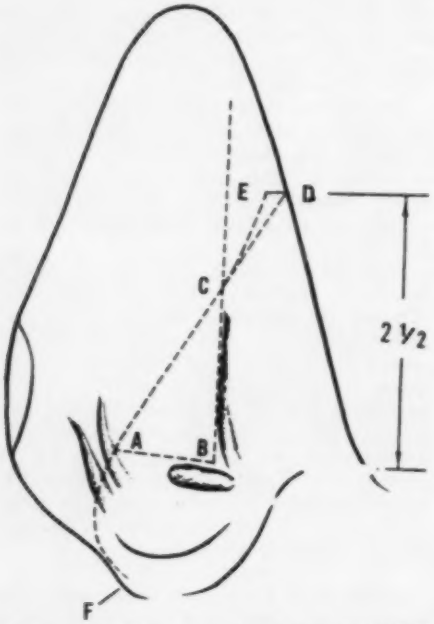

Fig. 1—To simplify ear cropping, a triangle (A-B-C) is drawn on the dog's ear, following the natural landmarks of the ear.

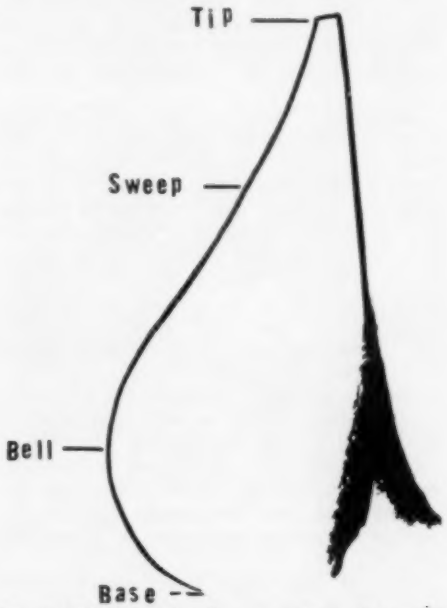

Fig. 2—Using the triangle as a pattern, the ear of the dog is cut from the base to the tip.

PRELIMINARY PROCEDURE

The length of the cropped ear is determined by the breed and size of the dog and, to some extent, by the wishes of the owner. In Boxers and Doberman Pinschers, about one third of the ear length is removed. In the Boston Terrier, the length is reduced little, if any.

A ruler is used for measuring, placing the end of the ruler at point B in the diagram (fig. 1). In the average Boxer pup, 8 weeks old, cropping is done at the 2.5 inch mark, starting at this point with a liberal cut from the anterior edge (fig. 1, line D-E). The tips of the ears are then brought together over the head and the cut in the first ear is used as the pattern for cropping the opposite ear.

Cutting the ears the proper width is the most important part of the procedure since it must be done in such a way as to not

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only make the ears attractive and uniform, but also to give the ears every possible chance to stand erect. There are certain natural landmarks on the ear which, when used properly, will serve as the necessary components of a triangle (fig. 1) used in determining where the ear is to be cut. When first attempting to use this method, clean the ear with ether and use a ballpoint pen to draw a triangle on the inner surface.

The outer surface of the ear has a groove which starts at the base (fig. 1, F). Starting at point F, follow this groove up to the point where it crosses the second ridge on the inner surface of the ear until point A of the triangle is reached. This point determines the width of the bell of the ear.

Point B is formed by the intersection of two ridges to form the imaginary right angle of the triangle. The horizontal ridge which lies anterior to point A is the base of the triangle. The vertical leg of the right angle is a small ridge which extends about one half the distance to the tip and parallel to the anterior edge of the ear. This vertical line should be extended to beyond the line D-E.

Figure 1 shows the base of the triangle (line A-B), the right angle (angle B), and the vertical leg (line B-E). The hypotenuse is formed by connecting points A and D, completing the triangle. Actually, line A-D will intersect line B-E about ¾ inch from point E or the tip of the cropped ear.

SURGICAL PROCEDURE

Sodium pentobarbital is routinely used for general anesthesia. Some veterinarians have reported using meperidine hydrochloride (Demerol*) intravenously and this technique has merit in that it effectively controls the extensive hemorrhage associated with ear cropping. It is recommended, however, that the veterinarian fully familiarize himself with the drastic blood pressure-reducing effect of this drug

*Produced by Winthrop Labs., Inc., New York, N. Y.

Fig. 3—The cropped ears are brought together over the dog's head and taped (above) to form an arch or are taped together (right). ➤

before attempting to use it intravenously if he wishes good results.

Infiltration of the skin along the incision site with a 1:10,000 solution of epinephrine hydrochloride before making the incision is a simple and effective means of controlling the hemorrhage. Another method of hemostasis is to "pull" the bleeding arteries after the incision is made.

Start the cut at point F on the outer groove at the base of the ear, cut directly to point A, and then follow line A-D. When approaching point C, begin to make a gradual sweep toward point E instead of point D. Point E will be about 0.5 cm. from the anterior edge of the ear. This will leave a slightly blunt tip to the ear (fig. 2). Ears that are cut to have a sharp point at the tip have a tendency to curl and will heal with more irregular scar tissue.

When cutting is finished, the layers of skin at the edge are drawn together and sutured, avoiding the cartilage, using interrupted stitches of 0, 00, or 000 plain catgut. The sutures start about 2 cm. below the tip and the skin is pulled taut as the base is approached. This will leave a small amount of loose skin which is drawn together at the base of the ear. If sutures are omitted, much more exacting aftercare is required.

AFTERCARE

The first day following surgery, the tips of the ears are brought together over the head and taped in place with a strip of ¼-inch tape on the dorsal and ventral surfaces (fig. 3A), or the ears are taped together over the head (fig. 3B).

The patient is released from the hospital on the second day following surgery, with instructions that it be isolated from litter mates and that care be taken to allow the edges of the ear to heal without injury.

We insist that the patient be returned about ten days following surgery or when the edges have completely healed. The tape is removed and the ears are rolled into a cone shape, taped loosely to hold them erect, but slanted somewhat laterally. The tape is left on the rolled ears for four days and then removed and the ears massaged.

Most ears will stand well by this time but a small percentage of them will need more training. A few ears will not stand even after long periods of taping. These become prospects for various forms of corrective surgery.

CONCLUSION

By using the natural landmarks of the canine ear as guides, the practitioner can develop a technique of ear cropping which will give results that are uniform, successful, and attractive.

Bovine Cesarotomy Case Reports

The author presents several interesting cases from over 1,000 cesarotomies performed at a clinic in Germany.

Case 1.—In May, 1950, a young cow, nine months' pregnant, was brought to the clinic in a state of paraplegia seven hours after an attempted forced extraction of the fetus. The day after cesarotomy and removal of the dead calf and fetal membranes, a complete prolapse of the uterus occurred. The uterus was soiled and some peritoneal fluid was escaping between the sutures but the incision was well closed. The uterus was returned to normal position and the cow recovered but did not again become pregnant.

Case 2.—In April, 1951, a 3-year-old heifer in labor four days, with the allantois burst for two days, was still rather lively. The cervix was open 10 cm., and one foot was protruding but the fetus was edematous and the uterus greatly involuted.

Upon laparotomy, 15 liters of clear, peritoneal liquid escaped. The pregnant horn could not be moved so it was incised intra-abdominally but, because of the condition of the fetus and the dry uterus, fetotomy was necessary. A second emphysematous calf was found and it and the fetal membranes were also removed.

In spite of fair tonicity of the uterus, there was serious hemorrhage from the caruncles, causing signs of anemia and dyspnea in the cow. She was given a 4-liter blood transfusion before the abdominal wall was sutured, and developed shock. She appeared to be dying but improved when given a cold water shower and stimulant treatments, so the operation was completed, taking a total of three hours. She was given another 4 liters of blood the next day and again developed a severe but brief shock. Recovery was gradual. She was again bred and calved normally.

Case 3.—In April, 1954, a heifer in labor developed a prolapse of the bladder which had existed six hours. The everted bladder was tight, edematous, and was believed to be filled with intestines. It could not be re-

duced. The calf was removed by cesarotomy but the intestinal loops could not be withdrawn from the everted bladder. After slaughter, the bladder was found to contain swollen portions of the colon.

Case 4.—In February, 1956, a heifer pregnant eight and one half months showed labor and abdominal pain. Upon laparotomy, 50 liters of sanguineous liquid and some blood clots escaped. The dead calf was removed by cesarotomy. The broad ligaments of the uterus were swollen and edematous and the uterus was found to have been twisted 720 degrees, two full revolutions. After surgery, there were signs of shock but the heifer responded to therapy and was released from the clinic in ten days.

Case 5.—In March, 1950, a multipara cow, supposedly nine months' pregnant, had developed, in the past two months, a huge abdomen until she could scarcely walk. Hydroallantois was suspected but only small quantities of liquid escaped after abdomino-uterine puncture. Upon hysterotomy, large tumor masses were found distending the right horn of the uterus and there was no fetus. The leiomyomas, up to 60 cm. in diameter, weighed a total of 280 kg. (616 lb.).—*M. Vandeplassche in Berl. u. Münch. tierärztl. Wchnschr. (March 15, 1958): 114.*

Progress in Bovine Obstetrics

Of 3,978 bovine cases at the Veterinary Obstetric Clinic, in Bern, the annual death rate fell from 25 per cent in 1871 to 6 per cent in 1957, chiefly due to the use of cesarotomy and antibiotics. The prognosis for the newborn also improved.—*W. Hoffman in Berl. u. Münch. tierärztl. Wchnschr. (March 15, 1958): 110.*

Swine Vibriosis (Dysentery).—Swine dysentery was common, in Poland, especially where the swine had been transported, feed had been changed, and sanitary conditions were bad. Peracute, acute, and chronic forms affected pigs 3 weeks to 5 months old. The chief lesions were hemorrhagic gastritis and fibronecrotic colitis.

Vibrio suis were isolated from the submucous membrane of the colon and from mesenteric lymph nodes. Agglutination and complement-fixation tests had no value for diagnosis.

The disease was controlled by isolation and treatment of the sick pigs, by strict sanitation, and by removal of predisposing factors. Streptomycin, chloromycetin, and sulfaguanidine were highly effective in therapy.—*Med. Wtryn. (Feb., 1958): 65.*

Therapy for Vibrio Fetus Infection in Bulls.—Infected bulls were given epidural anesthesia, then the protracted penis and preputial mucosa of 30 were treated locally with trypaflavin solution or ointment (as for Trichomonas infection) and 85 with a fat-free ointment containing streptomycin and terramycin. Later cultures from the preputial washings and from bred heifers were negative in all treated bulls in tests which continued from one to ten months. When applied carefully, this treatment is simple, inexpensive, and almost 100 per cent effective.—*Vlaams Diergeneesk. Tijdschr. (Jan., 1958): 1.*

Induced Vibriosis in a Heifer.—When *Vibrio fetus* was introduced into the vagina of a virgin heifer, the organism multiplied rapidly and infected the uterine cavity within seven days. After seven weeks, the uterus became free of infection without being treated, but the cervix and vagina remained infected for over eight months.—*Rec. méd. vét., d'Alfort (Feb., 1958): 97.*

Antibody Transfer from Mother to Child.—The incidence of certain diseases in 534 infants who were never given human milk and 534 who were breast fed was studied. Gastroenteritis occurred in 22 in the first and 21 in the second group; bronchopneumonia in 60 in the first group, 49 in the second group; and otitis media in 11 in each group. The incidence of measles, chickenpox, pertussis, and similar diseases was also about equal in both groups. This supports the view that, in man (unlike many animals), antibodies are transmitted from mother to offspring *in utero.*—*Brit. Med. J. (Jan. 25, 1958): 187.*

Dangers in Alimentary Lavage.—Laparotomies and necropsies of dogs, performed after thorough reverse washing of the digestive tract, indicated that lavage may produce injuries. When the tract was obstructed, hemorrhages were often found in the mesentery and subserosa of the bowels. Lavage should be used only when foreign bodies are in the stomach or are

not occluding the bowel. Many tapeworms and ascarids are not washed out of the bowels.—*Magyar allatorv. Lap. (Feb., 1958): 50.*

Puerperal Septicemia in Sows

Three days after parturition, in Holland, biopsies were taken from an inflamed mammary gland and from the uterus (via laparotomy), from 12 sows with puerperal septicemia and toxemia, for bacteriological and histological study. Similar tissues were taken at slaughter, three to five weeks later, of the 9 sows which survived.

Escherichia coli, often with cocci (with Klebsiella in 1 sow) were isolated, both times, from the uterus and mammary tissue in nearly all sows. In 30 per cent of the sows, there was a diffuse infiltration of the inflamed mammary tissue at the time of laparotomy and, at slaughter, the mammary tissue contained many small foci of pus. The findings in a normal control sow were negative.

For therapy, absolute rest and injections of oxytocin, penicillin, and streptomycin were recommended.—*Vlaams Diergeneesk. Tijdschr. (Feb., 1958): 42.*

Canine Pyometra with Perforation

Spontaneous perforation of the uterus occurred in 7 per cent of bitches with pyometra at the Giessen (Germany) Veterinary Clinic. It usually occurred in dogs when there was a hypoplasia of the muscular wall with a phlegmonous inflammatory infiltration of the muscles or with abscess formation.

The mortality after perforation was approximately 7 per cent even after hysterectomy. Prompt surgery is necessary but diagnosis is difficult without laparotomy.—*G. W. Rieck in Berl. u. Münch. tierärztl. Wchnschr. (March 15, 1958): 118.*

Treating Slow-Healing Wounds.—Nine patients, between 57 and 83 years of age, with ulcers of varying size and duration up to 20 years, were treated as follows, with recovery in 8 cases. A solution containing 10,000 units of human profibrinolysin, 150 units of hyaluronidase, and 5,000 units of streptokinase-streptodornase, each reconstituted in isotonic sodium chloride solution, was applied on wet compresses under a light bandage daily for a few days up to several weeks. The function of the streptokinase was to transform the inactive profibrinolysin into active enzyme fibrinolysin. No other medication was used. There were no side effects. —*J.Am.M.A. (April 5, 1958): 1791.*

Wound Healing in an Embryo.—Wounds made in chicken embryos after five to 12 days of incubation did not heal because of the marked incapacity of the epidermis to spread over the wound surface in spite of high mytotic activity at the wound margin. After day 12, epidermal regeneration was normal.—*Nature, 180, (1957): 854 (abstr. in Vet. Bull. (April, 1958): Item 1246).*

Sexual Behavior in the Cat

In studies of the neural and endocrine mechanisms in smaller mammals which could be used for laboratory tests of hormones, the female cat is an exception in that receptivity of the male is closely associated with the estrous cycle as determined by vaginal smears and by over 300 mating tests. Using males trained to attempt mating with any female released where the male had "territory rights," mating was allowed by only 1 of 142 females whose vaginal smears showed anestrus. In 300 attempts, mating was not accomplished with spayed females, confirming the dependence of estrous behavior on the presence of the ovaries.

In constrast, the female chimpanzee may accept the male at any time as, to a less extent, may the female rat and rabbit.—*Nature (Feb. 22, 1958): 567.*

Preventing Parturient Paresis with Vitamin B_3.—Three experiments, at the University of Utrecht, with 150 cows that had experienced parturient paresis at least once, indicated that the disease can often be prevented by the intravenous injection of 10 million I.U. of crystalline vitamin B_3 in a soluble form when given two to eight days prior to parturition.—*Tijdschr. Diergeneesk. (Feb., 15, 1958): 125.*

Infectious Agent from Bovine Coital Exanthema.—An infectious agent has been recovered, in cultures of embryonic cells, from the vaginal discharge of a cow with coital exanthema. It is thought to be a virus. Serological studies failed to reveal neutralizing antibodies in the serums of recovered animals.—*A. S. Greig et al. in Canad. J. Comp. Med. and Vet. Sci. (April, 1958): 119.*

A Comparison of Three Anthelmintic Preparations Against Nematodes of Sheep

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THE EXPERIMENT reported here was undertaken because of severe helminthiasis, during the summer of 1957, in a flock of about 200 breeding ewes and their lambs on irrigated pastures along the Little Big Horn River near Hardin, Mont. Gastrointestinal parasitism, suspected in July when severe diarrheas and deaths occurred, was confirmed by necropsies and fecal egg counts.

The lambs with heavy parasitism provided an exceptional opportunity to compare the action of three anthelmintics; a fine-particle purified phenothiazine, a commercial N.F. phenothiazine bolus, and tetrachlorethylene.

LITERATURE CITED

A number of workers have, in recent years, confirmed the finding that the anthelmintic activity of phenothiazine (PT) is variable, but samples composed of small particles are more efficient than samples of coarse particles.[6] However, no complete agreement has been reached on the optimum particle size or size range of PT to be used under all conditions.

Work done in Australia showed that a PT preparation having 100 per cent of the particles under 20μ reduced Trichostrongylus fecal egg counts by 90 per cent, and was more effective in removing worms than preparations containing larger particles.[4] In California,[1] a preparation of PT having 100 per cent of the particles under 15μ and a mean particle diameter of 1 to 2μ removed 93 per cent of small intestinal worms from sheep and was four times more effective in removing worms than a product containing particles averaging 40 to 50μ.

In another study, PT having particles averaging either 1 or 4μ was 98 per cent effective in removing worms, and PT averaging 10μ in particle size was 75 per cent effective. Since these preparations were relatively ineffective in removing Oesophagostomum and Chabertia, it was suggested[6] that some 30-μ particles should be included in order to remove those parasites in the large intestine and cecum.

Despite widespread use of tetrachlorethylene for removal of gastrointestinal nematodes of ruminants, its use is based primarily on the reduction in fecal egg counts and clinical improvement of the animals. Postmortem host examinations showed that even 5 cc. of tetrachlorethylene administered in a gelatin capsule was ineffective in removing helminths of sheep.[3]

MATERIALS AND METHODS

Forty parasitized lambs, weighing an average of about 40 lb., were selected at random from the flock and allotted to four groups of 10 lambs each. Two pretreatment fecal egg counts for Trichostrongylus and Nematodirus (table 1) indicated that the degree of parasitism for the four groups was comparable.

TABLE 1—Average Eggs Per Gram From Two Pretreatment Fecal Examinations on Lambs

Parasite	Control	Purified PT (5μ)	Commercial PT	Tetrachlorethylene
Trichostrongylus	2,576	2,522	2,604	2,482
Nematodirus	224	297	171	187

Two lambs died before treatment. The remaining lambs were treated on Aug. 17, 1957, as follows: group 1—untreated; group 2—12.5 Gm. of fine-particle (5μ av.) purified PT as a drench;* group 3—12.5 Gm. of a PT N.F. bolus; group 4—2.5 cc. of tetrachlorethylene in a gelatin capsule.

The lambs were killed in two lots four and five days after treatment. Abomasums and small intestines were removed and total worm collections made by passing their contents through a 40-mesh screen. Nematodes were preserved in 2 per cent formalin-glycerol for subsequent counting and identification. Total counts were made by an aliquot method and all worms in the aliquot were identified as to genus. Thirty worms were removed

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Paper No. 424, journal series, Montana Veterinary Research Laboratory (Montana Experiment Station and Livestock Sanitary Board cooperating), Agricultural Experiment Station, Montana State College, Bozeman.

The authors thank Dr. H. A. Brosz, Hardin, Mont., for use of his veterinary hospital and for his cooperation during this experiment.

*A special preparation provided by the Dow Chemical Company, Midland, Mich.

TABLE 2—Post-treatment Worm Counts and Percentage Removal of Worms

		Treatment			
		Control	Purified PT (5μ)	Commercial PT	Tetrachlor-ethylene
Location	No. of lambs	9	10	10	9
	Parasite species				
Abomasum	Ostertagia	1,982	68*	1,152	1,194
	circumcincta	.....	97%**	42%	40%
	Haemonchus	115	0	0	111
	contortus	.....	100%	100%	4%
	Trichostrongylus	393	28	16	638
	axei	.....	93%	95%	0%
	Total	2,490	96	1,168	1,939
		.....	96%	53%	22%
Small intestine	Trichostrongylus	8,825	782	2,587	4,943
	colubriformis	.....	91%	71%	44%
	Nematodirus	2,629	2	331	2791
	spathiger	.....	100%	87%	0%
	Total	11,454	784	2,918	7,734
		.....	93%	74%	32%
Abomasum and small intestine	Total for all species	13,944	880	4,086	9,677
		.....	94%	71%	31%

*Average number of worms at postmortem examination; **percentage removal as compared to untreated control animals.

from each sample, when possible, and identified as to species.

RESULTS

Compared to the untreated lambs, the greatest removal (94%) of nematodes occurred in the group given fine-particle, purified PT; the commercial PT bolus was fairly efficient (71%), and tetrachlorethylene was least efficient (31%) (table 2).

The fine-particle, purified PT was efficient in removing all the species of nematodes encountered in the lambs. The PT bolus was relatively poor against *Ostertagia circumcincta* (42%), and *Trichostrongylus colubriformis* (71%), but gave good removal of *Haemonchus contortus, Trichostrongylus axei,* and *Nematodirus spathiger.* Tetrachlorethylene failed to remove as many as 50 per cent of the worms of any species.

DISCUSSION AND CONCLUSIONS

The variability in efficiency of different samples of PT has been amply demonstrated,[7] but the reasons for this variation are not evident. Particle size of PT no doubt is one factor; but there must be other factors, as yet unknown.

Resistance to PT through long, continuous contact would appear to affect the susceptibility of the worms to the action of PT.[2] A report published in 1943 indicated a 90 per cent removal of Nematodirus by PT[3] but, more recently, it has been stated that "a satisfactory line of treatment is awaited" for Nematodirus.[5] In the experiment reported here, there was complete removal of Nematodirus with the fine-particle drench and 87 per cent removal with the commercial bolus. It is doubtful whether the worm population in these sheep had previously had significant contact with PT.

In general, it may be concluded from this work that the fine-particle purified PT drench is superior to the N.F. green bolus, and that both PT preparations are clearly more efficient than tetrachlorethylene. There appears to be little justification for the use of tetrachlorethylene in sheep.

SUMMARY

1) Forty lambs having naturally acquired, mixed nematode infections were allotted to four groups and treated as follows: (1) untreated controls; (2) 12.5 Gm. of fine-particle (5μ av.) purified phenothiazine (PT) in a drench; (3) 12.5 Gm. of PT N.F. in a bolus; and (4) 2.5 cc. of tetrachlorethylene.

2) Four and five days after treatment, the lambs were killed and their worm content indicated the anthelmintic effectiveness of fine-particle PT as 94 per cent, the PT bolus as 71 per cent, and tetrachlorethylene as 31 per cent.

3) The PT bolus was 87 to 100 per cent effective in removing *Haemonchus contortus, Trichostrongylus axei,* and *Nematodirus spathiger,* and 43 per cent and 71 per cent effective in removing *Ostertagia circumcincta* and *Trichostrongylus colubriformis,* respectively. The fine-particle PT was 91 to 100 per cent effective in remov-

ing all five species of worms. Tetrachlorethylene failed to remove as many as 50 per cent of the worms of any of the species.

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Vaccinating Sheep for Brucellosis

In 1954, 39,401 sheep in flocks infected with brucellosis, in Russia, were vaccinated with strain 19 of *Brucella abortus*. Some were revaccinated the following year. In 1956, cultures from fetuses of sheep vaccinated only once yielded Brucella in 19 of 206 cases while all of the fetuses from 20 sheep vaccinated twice were negative. Vaccinations should probably be done in summer and not at the time of insemination or during pregnancy.—*Vet. Bull. (April, 1958): Item 1004.*

Serological Tests of Semen for Brucellosis.—Brucella organisms were demonstrated in the semen from a bull which was positive to serological tests but showed no clinical signs of infection. Tube agglutination tests on the semen were negative. The ring test on semen was doubtful in a dilution of 1:10, and weak in 1:5.—*Vet Bull. (April, 1958): Item 990.*

An organism identified as *Brucella abortus* was isolated from abscesses in the testicles of a hare with bilateral orchitis.—*Vet. Bull. (April, 1958): Item 999.*

Pathology of African Swine Fever, a Comparison with Hog Cholera—An Abstract

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African swine fever is an acute, febrile, highly contagious, viral disease of swine in Africa that closely resembles hog cholera clinically and at necropsy. Since no effective treatment or means of immunizing against African swine fever has yet been found, it is imperative that the disease be recognized promptly should it enter the United States. Differentiation from hog cholera is largely dependent upon gross and microscopic lesions, supported by its immunological distinction.

This study was based on clinical and pathological examination of naturally occurring and experimental cases, employing two strains of each virus. Color photographs and motion pictures were made and these, along with the tissues collected, provide the material for study sets of each disease.*

A comparison of the salient clinical features is made (graphs 1 and 2) and the relative case incidence of the most significant lesions in each disease are shown (table 1). It is apparent from this table that most of the lesions which develop in these two diseases arise from the effects of the viruses on blood vessels and lymphoid cells.

The vascular changes in both diseases were most common in the capillaries and small arterioles, and these changes, histologically, were most often apparent in

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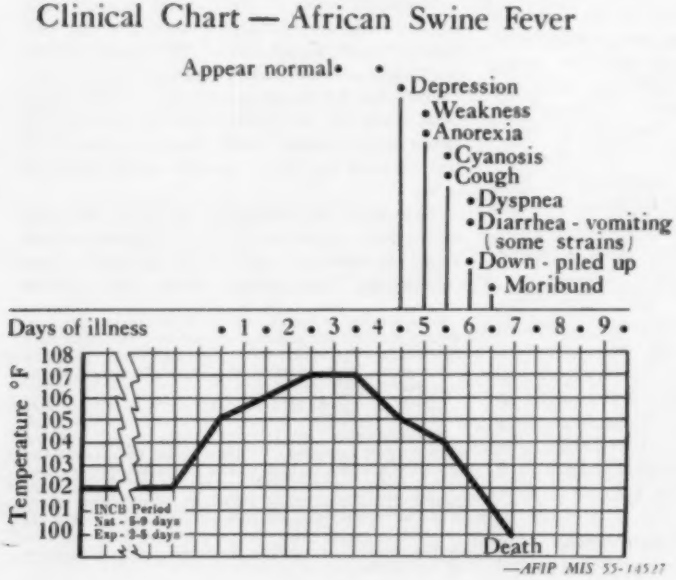
*Separate study sets of African swine fever and hog cholera, each consisting of 35 mm. color transparencies of the gross and microscopic lesions and tissue sections with commentaries, are available on loan from the Director, Armed Forces Institute of Pathology, Washington, D. C. A motion picture prepared jointly by the A.F.I.P. and the U.S.D.A., entitled "Hog Cholera and African Swine Fever, A Comparison" is available from the U. S. Department of Agriculture.

TABLE I—The Relative Case Incidence of the Most Significant Lesions in African Swine Fever and Hog Cholera*

Lesions	African swine fever	Hog cholera	Lesions	African swine fever	Hog cholera
Karyorrhexis of lymphocytes in lymphoid tissue and infiltrates	++++	.....	Skin, cyanosis	+++	+++
Capillary and arteriole, intimal damage	++++	++++	Heart, severe hemorrhage	+++	+
Capillary & arteriole, occlusion	++++	++++	Lung, petechiae and ecchymoses	+++	++++
Lymph node, peripheral hemorrhage	++++	++++	Lung, interlobular edema	+	.....
Lymph node, severe diffuse hemorrhage	+++	+	Kidney, cortical petechiae	++	++++
Leukopenia	++++	++++	Kidney, diffuse subcapsular or pelvic hemorrhage, or both	+	.....
Liver, lymphoid interlobular infiltrate with karyorrhexis	++++	.....	Urinary bladder, petechiae and congestion	++	+++
Central nervous system			Cecum and colon, inflammation, hemorrhage	+++	+++
Focal and perivascular hemorrhage	++++	+++	Cecum and colon, button ulcers (infarcts)	+	+++
Acute neuronal degeneration	++++	++++	Colon, severe submucosal congestion	+++	+
Perivascular and intramural infiltration	+++	++++	Excessive pleural, peritoneal, and pericardial fluids	+++	+++
Karyorrhexis, of lymphocytic infiltrate	+++	.....	Gallbladder, congestion and edema	+	.....
Meningeal congestion, lymphoid infiltrate	+++	+++	Splenic infarcts	+	++

*Each "+" represents a case incidence of about 25 per cent.

vessels of skin and lymphoid tissue. In areas of the skin presenting gross hemorrhage and cyanosis, capillaries were congested and many were partially or entirely occluded by dense, acellular, amorphous, eosinophilic masses (fig. 1) which were finely granular or slightly fibrillar and often adherent to the endothelium. Occasionally, leukocytes and erythrocytes were trapped in the masses, and endothelioid cells (but not fibroblasts) were present. The exact nature of this material is not clear but it resembles a platelet thrombus. It gives a positive periodic acid-Schiff (PAS) reaction (fig. 2). The walls of the capillaries often appeared slightly thickened, hazy, and pale-staining. At first, PAS-positive material accumulated just beneath the endothelium or elsewhere in the vessel wall and tended to form long hyaline



Graph 1—Diagrammatic representation of the sequence of clinical signs in African swine fever.

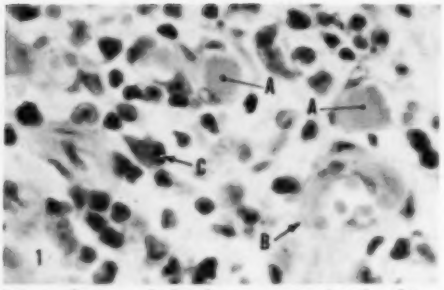


Fig. 1—Section of skin from an animal with African swine fever, showing (A) amorphous eosinophilic masses within capillaries, and (B) thickened hyaline capillary wall. The dark-staining cells (C) are eosinophils. H & E stain; x 800.

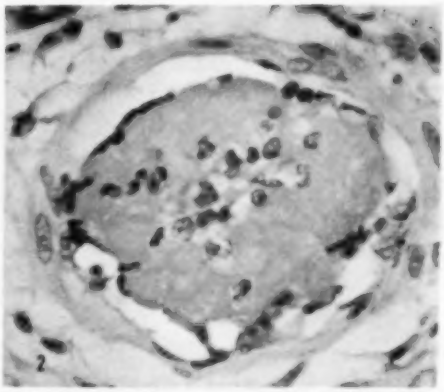


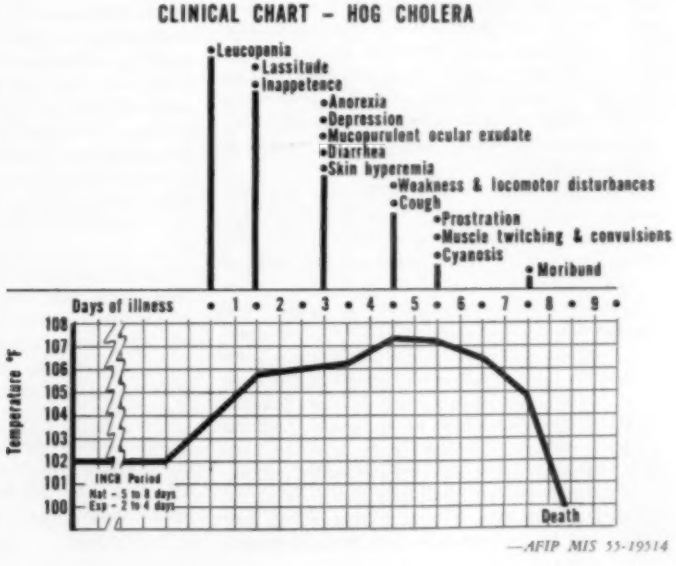
Fig. 2—Section of skin from an animal with hog cholera, showing occlusion of a capillary by a PAS-positive mass. Similar changes may occur in African swine fever. Endothelium covers the surface of the occluding mass and lines the vessel, and there are endotheliod cells within the mass. PAS stain; x 480.

strands. Later, much of the wall was replaced by PAS-positive material. The endothelium was frequently swollen but rarely hyperplastic. These same changes occurred in the capillaries of lymphoid tissues and were most severe adjacent to areas of severe lymphoid necrosis (fig. 3). The walls of such vessels were often thickened and pale-staining, and the lumens were obliterated or occluded.

These vascular changes indirectly accounted for the cyanosis, edema, and infarction. Infarction was primarily responsible for the necrosis in the tonsils and mucosa of the stomach, cecum, and colon (button ulcers).

The vascular changes in African swine fever and hog cholera are of the same general character, but the edema and hemorrhages in African swine fever are usually more diffuse and severe. Infarction is often more extensive terminally in hog cholera because of its longer course.

In African swine fever, the effect of the



Graph 2—Diagrammatic representation of the sequence of clinical signs in hog cholera.

—AFIP MIS 55-19514

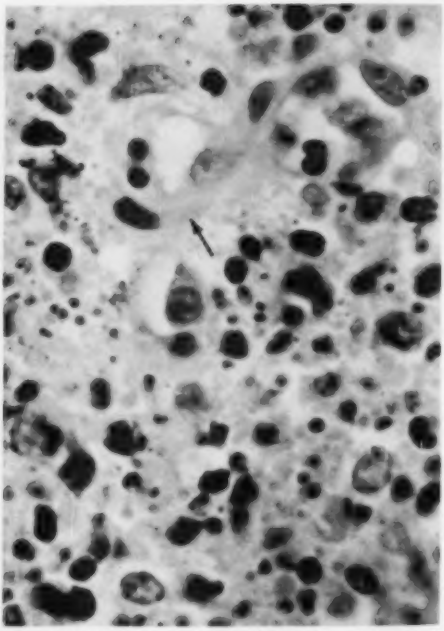


Fig. 3—Section of a lymph node from an animal with African swine fever, showing severe pyknosis and karyorrhexis of lymphoid cells and thickened, pale-staining capillary wall (arrow). H & E stain; x 930.

virus on lymphocytes is severe and their destruction with karyorrhexis is striking and characteristic. Karyorrhexis is readily apparent in lymphocytic infiltrations in the liver and brain, or wherever they are observed, as well as in the lymphoid tissue that is normally present (fig. 3). Depletion of lymphocytes also occurs in hog cholera, but fragmentation of their nuclei is not a typical feature.

The histological changes in African swine fever are more consistently significant for differential diagnosis than the gross lesions. Typical microscopic lesions in the lymph nodes, spleen, brain, and large intestine are diagnostic in an otherwise compatible case. A distinction from cholera can be confirmed by challenging pigs known to be cholera-immune with the suspected virus or, less desirably, by demonstrating the inability of anti-hog cholera immune serum of known high titer to protect test pigs against the suspected virus.

The only animal suitable for diagnostic inoculation is the domestic pig. When the diagnosis is to be made in a region where the disease has not occurred previously, the combined use of all methods in arriving at a definite diagnosis is recommended.

Pityriasis Rosea in Pigs

In an inbred line of pigs, in Germany, 13 of 18 litters had pityriasis rosea (false ringworm). When a new boar was used, only four of ten litters were affected. When a boar with this inbreeding was used on other sows, only one of 13 litters was affected. When the latter boar was crossed with a sow, from another breeding line, which had been affected with pityriasis rosea, all five of her litters were affected.

It is concluded that there is a hereditary predisposition for the disease.—*Vet. Bull. (April, 1958): Item 1199.*

Pasteurellosis in Swine

An acute infectious disease of swine, in Germany, was diagnosed as pasteurellosis. It had a tendency to spread but not to become enzootic. Brownish swellings in the throat were typical. There was pharyngitis and a phlegmon of the swollen peripharyngeal tissues, including the salivary glands and local lymph nodes. The swollen tonsils showed no diphtheroid or necrotizing processes; this may be of importance in differentiating this lesion from pharyngeal anthrax in swine.

Those which died after 36 hours of illness showed edema of the lungs, while those which died on the third day had a necrotizing bronchopneumonia throughout the lungs as well as a diffuse pleuritis and pericarditis.

The organism was sensitive to a penicillin-streptomycin combination, the use of which prevented a considerable economic loss when given early.—*H. J. Heidrich et al. in Der prakt. Tierarzt (May 1, 1958): 135.*

Listeriosis in Trout.—In the winter of 1956, in Romania, trout fed with the meat of a donkey developed apathy, brief periods of agitation, blackening of the integument, blindness, and inappetence. *Listeria monocytogenes* was recovered from the trout. Rabbits and guinea pigs inoculated with the organism developed keratitis and ophthalmia but pigeons and carp were not affected.—*N. Stamatin et al. in Yearbook, Inst. Anim. Path. and Hyg., Bucharest, 7, (1957): 180.*

Recent Experiences with Therapeutic Agents in Veterinary Dermatology

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NEW METHODS for the effective treatment of pyogenic infections in dogs and ringworm in dogs, cats, and horses are reported.

PYOGENIC STAPHYLOCOCCIC DERMATITIS

The effective treatment of pyogenic infections of the skin has been difficult, particularly in the last two years, in man as well as in animals. Short-haired dogs such as Boxers, Dachshunds, Labrador Retrievers, and Terriers, appear to be particularly susceptible. They are affected with severe skin conditions characterized by the formation of boils, acne, furunculosis, and superficial or deep, undermined pyogenic skin lesions.

In the majority of such cases, *Staphylococcus (Micrococcus) pyogenes* var. *aureus* has been isolated from both man and animals. Sensitivity tests revealed that these pyogenic strains of Micrococcus have been resistant to various antibiotics, including penicillin, streptomycin, neomycin, chlortetracycline (Aureomycin*), and others. Only novobiocin, erythromycin, and chloramphenicol have shown some degree of efficacy.

Because of the increasing number of resistant strains observed, our attention was focused on one of the newer synthetic organic compounds which is generically known as triocil (Hexetidine**). It possesses a broad spectrum of activity against bacteria and fungi.[4] Its effectiveness is believed to be due to interference with the metabolic processes necessary for the growth of pathogenic organisms.

As a topical ointment, it was found to be especially valuable in acute cases of superficial pyogenic dermatitis which developed as a secondary micrococcic infection in various areas damaged primarily by scratching or biting.

The deep form of pyogenic dermatitis, particularly the undermined form associated with acne and furunculosis, has always been difficult to treat, even when antibiotics are administered with enzymatic local or systemic therapy.

These stubborn pyogenic conditions have been considered systemic infections appearing as "boils" or "undermined" cutaneous lesions which discharge a purulent or sanguineous fluid from fistulous tracts. As a rule, these lesions develop in a symmetrical manner on the outer sides of the extremities, on the lower or upper lips, between the toes, and commonly on other areas of the body surface.

The lesions may appear to respond to the combined antibiotic-enzymatic treatment, only to have new lesions start in other areas after several days. In all animals in which bacteriological tests were made, *Micrococcus pyogenes* var. *aureus* was isolated. Sensitivity tests, *in vitro*, with the antibiotics referred to above, demonstrated some bacteriostatic activity. However, when used *in vivo*, results were poor. Apparently, the artificial culture medium, used for the sensitivity tests *in vitro*, offers different conditions for growth of the pathogenic microorganism than in the infected animal.

Treatment and Results.—Therefore, instead of the bacteriostatic treatment with antibiotics, we initiated therapy with a systemic bactericidal agent, a bismuth preparation, sodium bismuth triglycollamate (Bistrimate†), and triocil spray. The bismuth preparation has been used successfully in man for the systemic treatment of syphilis and acne.[1,2] It is administered to dogs orally as tablets containing 75 mg. of bismuth—4 tablets daily in divided doses for four days, 2 tablets daily for four days, then 1 tablet daily for eight to 12 days. This dosage causes no signs of toxicosis or discomfort in an average size dog.

The triocil spray (bis-1, 3-*beta*-ethylhexyl-5-methyl-5 amino hexahydropyrimidine), 0.5 per cent of the active ingredient in an alcohol-acetone base, demonstrated bacteri-

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*Aureomycin is produced by American Cyanamid Co., New York, N.Y.

**Hexetidine is a product of Warner Chilcott Laboratories, Morris Plains, N.J., and is available as an ointment, detergent solution, and a spray.

†Bistrimate was supplied by Carroll Dunham Smith Pharmacal Co., New Brunswick, N.J.

Fig. 1—Ringworm in a Boxer, caused by Microsporum gypseum.

cidal effects both *in vitro* and *in vivo*. It penetrated the affected skin without causing irritation. It was applied twice daily for six to eight days. When the lesions were on the lower parts of the extremities and showed itchiness, it was necessary to protect the affected area with a gentle bandage fixed with adhesive tape.

We have treated 42 animals affected with superficial, deep, and undermined pyogenic dermatitis, some of which were considered incurable and assigned for euthanasia. Of the 42 dogs, 36 recovered completely within three to six weeks of treatment, 4 improved essentially, and 2 were euthanatized, due to age and poor condition. Response to treatment was noticed after several days. The suppuration and discharge from the fistulous tracts stopped and crusts covered the treated areas. The crusts were removed by the local application of an oily preparation.

For the final treatment of the skin we used a triocil solution, the detergent base of which permitted bathing of the whole body to remove scaliness from the healed skin and to disinfect the skin and haircoat. This is desirable because staphylococci can survive and maintain their virulence for a long time in a dry haircoat and in fine dry dust.

DERMATOMYCOSES

Dermatophytic infections of the skin are important in both human and veterinary medicine because of their mutual transmissibility. We have isolated the following six species of animal dermatophytes belonging to two main groups, Microsporum and Trichophyton: in dogs—*Microsporum canis, Microsporum gypseum*, and *Trichophyton mentagrophytes;* in cats—*M. canis;* in monkeys—*M. canis* and *M. gypseum;* in chinchillas—*M. canis* and *T. mentagrophytes;* in horses—*T. mentagrophytes* and *Trichophyton equinum;* in cattle—*Trichophyton verrucosum;* in poultry—*Trichophyton gallinae.*

There have been favorable reports on the use of a "tamed iodine" (Weladol‡) in the form of cream, ointment, or shampoo for dermatomycosis.[5] I have used another fungicidal preparation, a triocil spray, on dermatomycotic skin lesions in dogs (fig. 1), cats, and horses, caused by *M. canis, M. gypseum*, and *T. mentagrophytes.*

This spray was tested first *in vitro* on skin scrapings taken from affected areas and showing either fluorescence, on Wood's Lamp irradiation, or fungus elements microscopically. One spraying was sufficient to inhibit the growth of the live dermatophytes on the usual Sabouraud medium. The presence and vitality of the fungi was proved by the control culturing of unsprayed scrapings from the same skin area.

In another experiment, when the culture medium was sprayed before the scrapings

‡Weladol is produced by West Disinfecting Co., Long Island City, N. Y., and Pitman-Moore Co., Indianapolis, Ind.

with live dermatophytes were put on the medium, growth was not evident. Finally, 1 drop of the spray, dropped on the growing culture of *M. gypseum*, immediately inhibited growth of the fungus and, when a part of the treated culture was subcultured, it showed no vitality.

Treatment and Results.—The spray was then used for the topical treatment of 18 animals—11 dogs, 5 cats, and 2 horses with ringworm caused by *M. canis*, *M. gypseum*, and *T. mentagrophytes*. The spray was applied to the affected areas once a day for four to six days, depending on the severity of the lesions. All animals recovered from the lesions within two to three weeks but, in 6, new ringworm lesions developed on other areas as a result of infection prior to treatment of the first lesions.

Therefore, in the next animals treated, after the ringworm disappeared, the whole body surface was treated with the triocil solution by bathing. (The solution differs from the spray in that it has a detergent base which makes it possible to use it as a medicated bath.) No ringworm lesions have been noticed since. The spray apparently caused no discomfort and was effective, especially in the treatment of ringworm in cats and dogs when the ringworm lesions were caused by *M. canis*. Infections of this type were detected by Wood's Lamp irradiation. After three to four treatments with the spray, the fluorescence disappeared and the cutaneous lesions healed.

DISCUSSION AND SUMMARY

Of 42 dogs with superficial, deep, and undermined pyogenic dermatitis treated, 36 recovered completely within three to six weeks, 4 improved, and 2 were euthanatized because of age and poor condition. For the treatment of the superficial pyogenic dermatitis, only the topical treatment with triocil (Hexetidine) spray was used. For the deeper and undermined form of pyogenic dermatitis, oral treatment with a bismuth preparation (Bistrimate) and topical treatment with the triocil spray was effective, even in stubborn and long-lasting cases.

The spray was effective for ringworm in dogs, cats, and horses caused by *Microsporum canis*, *Microsporum gypseum*, and *Trichophyton mentagrophytes*. Of 18 animals treated (11 dogs, 5 cats, 2 horses), all recovered from ringworm lesions within two to three weeks. As final treatment, the entire body surface was bathed with the triocil solution to disinfect the skin and the haircoat, thus to avoid the development of other ringworm lesions.

Cats, particularly mature ones, are often carriers of the *M. canis* infection without showing skin lesions, thus being the main source of the dermatophytic infection for kittens and even for man. Therefore, it is necessary, when kittens have ringworm, to check the mature cats with Wood's Lamp and, if positive, to administer the necessary treatment.

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Lymphogranulomatosis in the Dog.—A female Boxer, 6 years old, had been developing multiple nodes in the skin for one year. Necropsy revealed lesions also in the lymph nodes and spleen. On the basis of the polymorphous giant cells which seemed to be inflammatory and proliferative, the lesions were considered to be lymphogranulomatosis, similar to that in man.—*H. Köhler in Monatsh. f. Vet.-med. (April 15, 1958): 230.*

Convulsions from Use of Chlorpromazine.—The epileptogenic effect of chlorpromazine was studied experimentally by comparing its effect on normal persons with that on individuals known to have latent convulsive tendencies.

Electroencephalograms showed that no changes were produced in normal persons but showed significant abnormality, as well as actual convulsive seizures, in susceptible persons even though they had no previous clinical history of seizures.—*J.Am.M.A. (April 19, 1958): 1986.*

The Incidence of Demodex Species on Skins of Apparently Normal Dogs

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THE MITES of Demodex sp. have been found in the skin of many mammalian hosts, and when they are associated with a dermatitis, the name "demodectic mange" has been designated for the lesions. At one time or another, many different types of skin lesions have been believed to be a result of Demodex infection. The presence of Demodex mites in the skin of apparently healthy animals raises the question as to the primary cause of this skin condition.[3,18]

The glands of the skin in the affected areas contain large numbers of the parasites, as many as 80,000 mites having been found in 1 sq. cm.[9] The glands and hair follicles at the center of a lesion are "overstuffed" with the parasites, while those at the periphery contain lesser numbers. In certain areas hair follicles may be completely destroyed.

In a brief review of available literature, it is evident that different and even conflicting views exist with reference to the Demodex mites and demodectic infections.

One author[11] believes demodectic mange can be transmitted congenitally and lists five reasons to substantiate this thesis. Another[6] theorized that the parasite may exist in the soil in certain areas.

The spread of Demodex to young pups nursing a carrier dam has been considered.[2] Mites have been found in a 6-week-old Dachshund pup of an affected mother,[13] yet infection was found in a litter of 9 pups from a mother that had never shown lesions.[17]

Mites have been considered normal inhabitants of the skin of dogs, which cause lesions only when some predisposing factor is present.[20] Others do not consider that the demodectic mite is a normal saprophyte.[4] An increasingly prevalent idea is that the presence of mites in the hair follicles is a result, and not the cause, of the condition termed demodectic mange.[16] In some treated animals the lesions apparently disappeared, but the parasites remained. In other dogs, the mites disappeared but alopecia remained.[10]

In one report, 9 of 91 apparently normal dogs were found to have mites in their skins. The appearance of lesions was thought to be due to the large number of mites present.[19] Large numbers of mites may act as foreign bodies, resulting in an increase in sebum.[7] The cells of the germinative bed of the sebaceous glands may also be irritated, thus producing lesions. There are indications that the prevalence of demodectic mange apparently follows the law of periodicity of other parasitic maladies or pests, and "rebellious dermatitis" may follow treatments that increase irritation of the skin.[8]

Several methods for demonstrating the mites have been described. The use of potassium hydroxide to separate mites from the skin debris has been advocated.[1,10,12] Sodium hydroxide has been used in isolating mites from lymph node tissue.[9] Mites were difficult to find in the skin of certain dogs treated with rotenone and oil.[15] In some animals where skin examinations were negative, mites were often found on fecal examination.

Because of statements in the literature concerning the presence of the Demodex sp. mites in the skin of clinically healthy animals, the following investigations were undertaken.

MATERIALS AND METHODS

Sections of skin were taken from 93 dogs at necropsy, at Texas A. & M. College, from April, 1957, to January, 1958. None of these dogs showed visible skin lesions. Their ages varied from 2 weeks to 11 years and the dogs were of several breeds, with 50 per cent being of mixed breeding.

The skin specimens were 0.5 cm. in diameter and were taken from the upper eyelid and temporal regions of each dog. They were placed in tubes containing 10 per cent potassium hydroxide. The tubes

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This project was supported by Grant E-1206 from the National Institutes of Health, U. S. Public Health Service, Washington, D. C.

were heated almost to boiling and then set aside at room temperature for 12 to 18 hours before being examined.

The tubes were then centrifuged at 1,500 r.p.m. for five minutes and the sediment was examined microscopically.

RESULTS AND DISCUSSION

Demodex folliculorum was found in the sediment of five (5.4%) of the 93 specimens. Although this finding is not unique, its significance substantiates the claim that demodectic mites can be found on healthy animals and that follicular mange is a complex condition.

The difficulty in finding the mites in the skin of dogs that have been treated to any extent may support the theory that the mites are easily killed, at least in the adult stage. Recurrence of the disease after disappearance of the visible lesions also supports this theory. Four of the 5 affected dogs were over 3 years old, indicating that although follicular mange is found mostly in dogs under 2 years of age, older dogs may have the mites in their skin and not exhibit lesions.

The presence of fungal, bacterial, or viral organisms may be necessary for the development of this complex skin condition. Certain predisposing factors, such as hormonal imbalance, malnutrition, and concurrent disease, apparently influences the susceptibility of the animals and the severity of the disease. The failure of attempts to artificially reproduce the disease or to raise the mite on artificial mediums supports this opinion. Until such time when the skin of dogs can be an optimum site for the mutual development of these parasites, it will not exhibit any lesions.

The number of apparently normal dogs in which mites were found is probably the minimum rather than the maximum number affected, since only a small portion of skin from each animal was examined.

SUMMARY

Demodex sp. was found in the skin of 5 of 93 clinically normal dogs. Some possible causes of follicular mange complex are discussed.

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Sulfamethoxypyridazine for Urinary Infections.—Since many patients are refractory to treatment with antibacterial drugs, a new sulfonamide, sulfamethoxypyridazine, seems suitable for long term chemotherapy. In 48 human patients treated for an average of 35 days, the usual dosage was 2.5 Gm. for the first dose, followed by 0.5 Gm. daily. The results were favorable in 80 per cent with acute infections and 24 per cent with chronic infections. —*J.Am.M.A. (March 15, 1958): 1386.*

A Combination Restraint Table and Cassette Holder for Routine Chest Radiography in Unanesthetized Monkeys

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We have previously demonstrated[1] that taking routine chest radiographs of monkeys is a valuable aid to the tuberculin test in detecting pulmonary tuberculosis.

A method of restraining monkeys was devised in order to handle large groups of animals speedily and easily without exposing the attendants to dangerous levels of x rays.

From the Animal Hospital, Chicago Professional Colleges, University of Illinois, Chicago.

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MATERIALS AND METHODS

A small animal operating table was constructed of hardwood and brass screws with three cassette holding slots (fig. 1).

The unanesthetized animal is tied to the table with leather thongs in the dorsoventral position, facing the operator. The animal's curiosity causes it to keep its head in line with the spine and prevent unwanted rotation of the chest. To prevent the scapulae from partially obscuring the cranial end of the chest cavity, the forelimbs are extended to the maximum without causing discomfort to the animal.

Any size cassette up to 13¼ inches in its smallest dimension could be used by fitting a 13¼-inch cassette into one slot or by placing a smaller cassette upon an empty 13¼-inch cassette in the slot (fig. 2).

The upper slot is used for small or shallow-chested animals (monkeys); the lower slot for deep-chested animals (dogs) (fig. 3).

Satisfactory radiographs were obtained

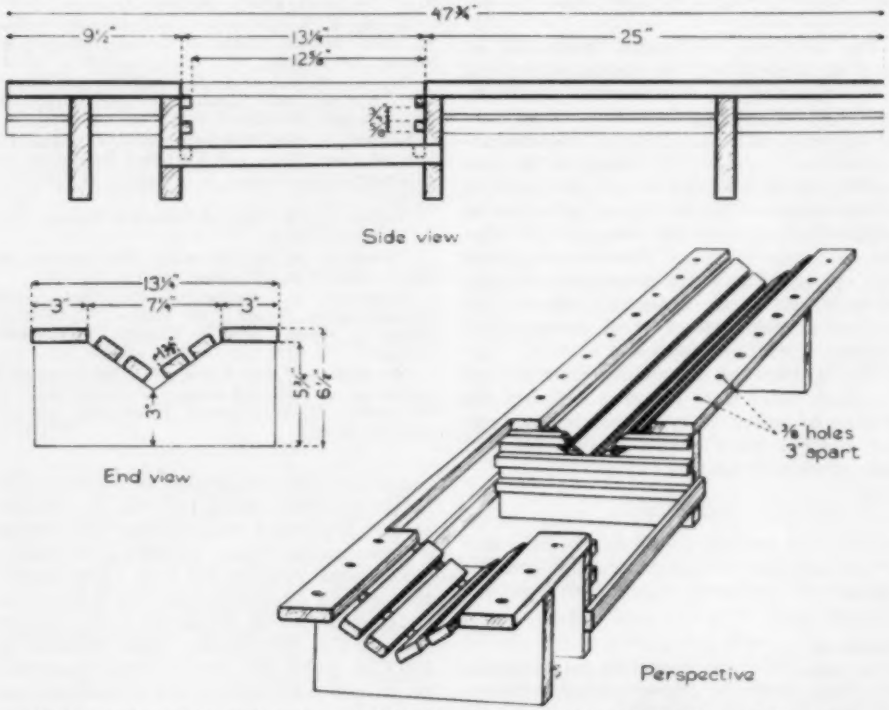

Fig. 1—Drawing of the combination restraint table and cassette holder showing the exact dimensions of all important components.

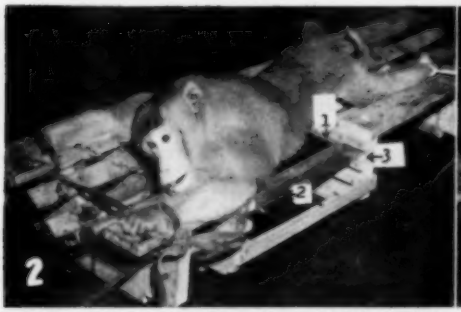

Fig. 2—Restraint table and cassette holder with monkey tied in approximate position for chest x ray, small cassette (1) placed upon large cassette (2) in slot (3).

Fig. 3—Restraint table and cassette holder with dog tied in position for chest x ray. Cassette (1) is placed in lowest slot (2) with those deeper-chested animals.

of monkeys weighing 10 to 25 lb. with the following settings: 48 kv.p., 1/15 to 1/10 sec., 100 ma., with a 36-inch target distance, using double screen cassettes. The films were sight developed.

A Microfilaria Contaminating a Monkey Kidney Cell Suspension

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The occurrence of filarid parasites in primates is well documented,[1,2,4,5] and the occurrence of filarid worms of the genus Dirofilaria in the African green monkey, *Cercopithecus aethiops*, has also been discussed.[6]

This report concerns the observation of microfilariae in a suspension of kidney epithelial cells obtained from an adult monkey of this species for use in routine tissue culture studies. Unfortunately, by the time the parasite was discovered, the carcass was destroyed and no search could be made for the adult worm. The morphology of the microfilarid, however, was similar to that described as typical of the genus Dirofilaria.[6]

The kidneys from an anesthetized monkey were removed from the capsule and washed in Hank's balanced salt solution (BSS). The kidney was then halved transversely and the cortical tissue removed. The cortical material was cut into cubes measuring approximately 0.5 cm. and again washed in Hank's BSS. The cortical material was trypsinized according to the method described[3] and the final suspension was centrifuged at 500 r.p.m. for 30 minutes.

The sedimented cells were resuspended in a 0.5 per cent lactalbumin hydrolysate medium and diluted, and trypan blue was added to stain dead cells. Cell counts were made in a Levy counting chamber.

Examination of the first sample and several others taken from the stock suspension revealed numerous actively motile microfilariae (fig. 1).

It is significant that the microfilariae were present in large numbers in spite of several washings and trypsinizations of the cortical tissue. It is improbable, because of this dilution factor, that the tissue was

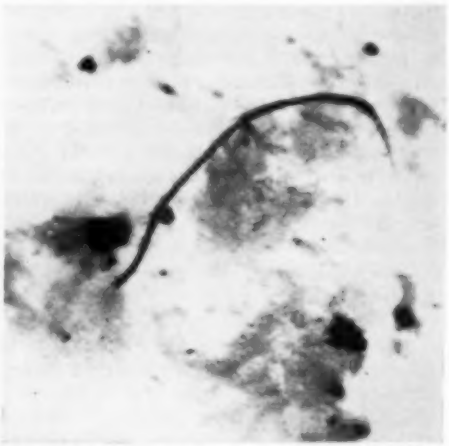

Fig. 1—Photomicrograph of microfilariae identified in a trypsinized monkey kidney cell preparation. Giemsa's stain; x 430.

From the Biological Development Department, Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa.

contaminated by peripheral blood-borne microfilarids, but rather it seems probable that the parasite was resident in the kidney tissues.

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Leukosis of Cattle

Because of the great increase in bovine leukosis, in Sweden, in the past decade, the disease was investigated by a research group. Analysis of the tumorous material showed it to be similar to that in fowl leukosis.

As in other countries, the disease was restricted to certain regions. Two theories as to etiological factors were advanced: (1) It occurred chiefly in areas with a high incidence of piroplasmosis, and had increased after vaccination against that disease had become general (the material for production of piroplasmosis vaccine originated from animals in a section where leukosis was prevalent); (2) leukosis occurred where the climate was dry, the soil poor in phosphate, and where there was an abundance of Ranunculus (buttercup type plant), which has a toxin with a carcinogenic effect. Neither theory has been proved.—*A. Hjarre Monatsh. f. Vet.-med. (March 15, 1958): 164.*

Tuberculous Lesions in Foxes.—In 19 tuberculous silver foxes, in Europe, lesions were found in nearly all organs but particularly in the lungs, liver, and spleen. Most tissues were icteric. In the liver, histiocytes in capillaries increased so as to cause destruction and atrophy of parenchyma cells, and the endothelial cells of the capillaries phagocytized tubercle bacilli as did histiocytes from the lining of the larger vessels. Reticular and sinus cells were activated in the spleen. In no organ was there formation of specific granulation tissue.—*Vet. Bull. (April, 1958): Item 976.*

Atrophic Rhinitis in Swine

Sows and their pigs, all showing signs of atrophic rhinitis, were studied. Antibodies against viruses of the psittacosis-lymphogranuloma group were found in the serums of the sows, and both intra- and extracellular corpuscles, with morphology and staining properties of Rickettsia, were found in the organs of both the sows and the pigs. The initial lesions were found in the nerves of the turbinate bones. The nerve lesions could have been caused by various etiological agents.

In certain cases, the disease apparently was transmitted in the uterus. This infection could cause abortions and stillbirths. The interference with growth of affected pigs is probably due to hepatic lesions, involving vitamin A deficiency.—*A. Brion in Rec. méd. vét. (Dec., 1957): 1035.*

Infection of Swine with Nematodes of Ruminants.—*Trichostrongylus colubriformis* and *Trichostrongylus axie* matured in normal young pigs, whereas *Ostertagia ostertagi* developed only in weak pigs. *Dictyocaulus viviparus* and Cooperia spp. did not develop in either weak or healthy pigs. —*Vet. Bull. (April, 1958): Item 1122.*

Pancreatic Carcinoma in Carnivores.—Of 5 cases of carcinoma in the pancreas of dogs and cats, 4 formed metastases in the liver and other organs. There was obstructive jaundice in 4 cases and ascites in 2. Histogenetically, the tissue was pancreatic epithelium.—*H. Stunzi et al. in Monatsh. f. Vet.-med. (April 15, 1958): 251.*

Treating Canine Toxoplasmosis

When the head of 1 of 2 adult Pointers, which had died after showing signs of meningitis, was sent to the Pasteur Institute, in France, the infection was diagnosed as toxoplasmosis.

When a 6-year-old Setter in the same kennel developed signs of encephalitis, hyperthermia, and paraplegia, it was treated

with sulfonamides without effect but recovered after eight days of treatment with carbomycin (1.0 Gm./day in divided doses for 2 days, then 0.5 Gm./day for 6 days). Dogs with distemper or hard pad disease similarly treated showed no benefit.—*M. A. Lebeau in Bull. Acad. vét. France (Nov., 1957): 475.*

Vertebral Ankylosis in the Dog

Of 6 dogs with stiffening of the spinal column, 5 were males. The ossification had started in the ligamentous apparatus, the most severely affected being the longitudinal ventral ligament of the last thoracic and lumbar vertebra. The exostoses varied from osteophytes to bony adhesions.

Changes in the dorsal ligament were less evident but in 1 dog there was an ankylosis of the articular processes between the tenth and eleventh thoracic vertebrae. Changes in the prostate were considered to be an etiological factor.—*J. Martin in Monatsh. f. Vet.-med. (April 15, 1958): 237.*

Intervertebral Disk Disease in Cats

The cat has been considered to be less susceptible to intervertebral disk diseases than are dogs and man. However, examination of 100 adult cats showed dorsal protrusions in 26, with 91 disks being affected. In some, the protruding mass perforated the annulus fibrosus; in others, there was a bulge without perforation.

In 50 cats examined for other abnormalities of the disks, ventral protrusions were found in 8, a lateral protrusion in 1, calcification in 3, and brown degeneration in 15.

General disorganization (sometimes almost complete destruction) of a total of 178 disks was found in 27 affected cats. While no disks were exempt, this degeneration was most common from the second to the tenth thoracic disk, whereas dorsal protrusions were few or absent in this region, possibly due to the support of ligaments.

In the dog, the highest incidence of both dorsal protrusion and degeneration occur in the last three thoracic disks where there is maximum mobility and curvature. However, since there are few diseased disks in this area in the cat, where there is also maximum mobility and curvature, it would seem that these are not important causes of disk diseases in this species. While this kyphosis may appear similar in the dog and cat, the reasons for it may differ. The extent to which it depends upon wedge-shaped disks and vertebrae should be studied.

The finding of disk diseases in the cat as well as the dog casts more doubt on the idea that man's susceptibility to disk protrusions is simply due to his upright posture.—*A. S. King and R. N. Smith (Dept. of Veterinary Anatomy, University of Bristol) in Nature (Feb. 22, 1958): 568.*

Brain Changes in Dogs with Induced Virus Hepatitis.—The brains of 13 dogs which died four to seven days after artificial infection with the virus of contagious hepatitis showed histopathological changes essentially similar to those in fox encephalitis. These included swollen endothelial cells of the vessels, inclusion bodies in their nuclei, and perivascular infiltration and hemorrhage. There was no relationship between the extent of the changes in the brain and in the liver.—*J. Sályi et al. in Monatsh. f. Vet.-med. (April 15, 1958): 242.*

Bovine Shock from Equine Serum

Shock, with 7 fatalities, occurred in 33 cattle immediately after parenteral injection of anti-tetanus serum made from horses.

In 32 animals, the serum was given subcutaneously or intramuscularly with the first sign of shock appearing in five to 20 minutes. In 1 case, when 3 ml. of serum was accidentally injected intraperitoneally, shock occurred in one minute. In 30 of the animals, including 6 of 7 that died, equine serum had not been given previously. The animals died within ten to 30 minutes after the first signs of shock.

Of the 9 animals given atropine intravenously and 30 to 40 ml. of 50 per cent Lugol's solution in water subcutaneously, there was quick improvement and 8 recovered, compared with 9 recoveries in 15 other serious cases not treated or treated by other methods.—*F. Buhner in Berl. u. Münch. tierärztl. Wchnschr. (April 15, 1958): 141.*

Blackleg in Swine.—An enzootic of spontaneous infection with *Clostridium chauvoei*, in Italy, resulted in the death of 15 of 34 swine in two days. The most constant lesion was edema of the pharyngeal region. —*Arch. Vet. Ital. (Feb., 1955): 57.*

The Effect of Vitamin A Supplement on the Incidence of Pneumonia in Lambs

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PNEUMONIA COMPLEXES in lambs cause a great loss to the sheep industry through deaths, poor weight gain, and failure to pass the required meat inspections. Various antibiotics have been used in the feed in an effort to control pneumonia in lambs,[3,4] without significantly altering the loss due to pneumonia.

The purpose of this investigation was to determine the possible effect of vitamin A supplement on the incidence of pneumonia in lambs during the nursing period.

MATERIALS AND METHODS

Forty-five Shropshire lambs, 4 to 7 weeks old, from the station flock, were used in this study. Vitamin A was not given before this age since pneumonia in lambs was not in evidence until they were older than this.[1] The lambs were allotted to two comparable groups. Each of the first group of 22 lambs was given 10,000 U.S.P. units of vitamin A, orally once a week, from May 1, 1957, until weaning time, August 15. The second group of 23 untreated lambs served as controls.

Vitamin A level was determined in the blood of 21 lambs pooled in seven samples prior to using the supplement. Plasma vitamin A was determined colorimetrically.[2] The lambs were kept in the barn from birth until May 6, when they were turned out to blue grass pasture.

Alfalfa hay, forage crop silage, and shelled corn and oats in equal parts were available, in creeps, to the young lambs during the vitamin A treatment. After weaning, they were fed whole shelled corn in the barn. The lambs were weighed every two weeks. The average daily weight gain for each group was calculated for the first 30 days of age and at weaning time.

They were given two doses of 10 Gm. of phenothiazine as a parasiticide on August 15 and September 10. They were observed for evidence of pneumonia and their carcasses were graded at slaughter either on Dec. 17, 1957, or Jan. 10, 1958.

RESULTS AND DISCUSSION

The average blood level of vitamin A of the lambs before treatment was 12.5 μg. per 100 ml. of blood, with a range of 7.9 to 18.6 μg. One day after the first dose of vitamin A, a pooled sample from 3 lambs showed a rise to a level of 30.1 μg. per 100 ml. of blood. The number of lambs having pneumonic lesions and the average daily weight gains in both groups are shown (table 1).

There was no death loss due to pneumonia in either group. One treated lamb died at the end of the trial from suspected weed poisoning and, on necropsy, its lungs were essentially normal. Examination at slaughter revealed that 47 per cent of the 21 lambs given vitamin A showed pneumonic lesions, most of them mild and chronic in nature, and 5 per cent had pleural adhesions. The percentage of similar pulmonary involvement in the control group was 30.5, with 4.5 per cent having pleural adhesions. The incidence of pneumonic conditions in both groups was within the limits of what might be expected from earlier observations.[1,5]

There was little difference in the average daily weight gain in the two groups up to 30 days of age. For the treated lambs it was 0.372 lb. (range 0.211 to 0.490 lb.),

TABLE 1—Effect of Vitamin A Supplement on Incidence of Pneumonia and on Weight Gains in Lambs

Date examined	Group	No. of lambs	No. of pneumonic lesions	Average weight gains: Pretreatment 1 mo. (lb.)	Average weight gains: Post-treatment 4 mo. (lb.)
12/17/57	Vitamin A	11	6	0.358	0.278
12/17/57	Control	11	3	0.390	0.307
1/10/58	Vitamin A	10	4	0.385	0.315
1/10/58	Control	12	4	0.352	0.315
Total	Vitamin A	21	10	0.372	0.296
	Control	23	7	0.370	0.311

From the Ohio Agricultural Experiment Station, Wooster.
Prepared in cooperation with the American Association of Veterinary Nutritionists.
The authors thank Mr. C. Bradford and Mr. R. Stall of the Animal Science Department for their assistance.

and for the control lambs it was 0.370 lb. (range 0.137 to 0.537 lb). During the experimental period, the average weight gain for the treated lambs was 0.296 lb. (range 0.235 to 0.427 lb.), and for the controls 0.311 lb. (range 0.138 to 0.427 lb.) (table 1).

At slaughter, it was found that vitamin A did not affect the carcass grades. In the treated group, 6 lambs were graded "prime," 13 "choice," and 2 "good" while, in the control group, 9 lambs were "prime," 13 "choice," and only 1 lamb was graded "good."

SUMMARY AND CONCLUSIONS

Feeding vitamin A at levels of 10,000 U.S.P. units weekly for 16 weeks during the nursing period, beginning at 4 to 7 weeks of age, did not lower the incidence of pneumonia in lambs. Pulmonary lesions of equal severity were observed in both treated and control lambs.

Differences of consequence were not observed in weight gains or carcass grades in either the vitamin A supplemented group or the control group.

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Salt Tolerance of Animals

When sheep on similar rations were given rain water to drink, containing 0, 1.0, 1.5, or 2.0 per cent of salt (NaCl), there were no adverse effects from the 1.0 per cent level but 1.5 per cent was detrimental to some, and 2.0 per cent was detrimental to all 6 sheep in the group. The effects were: a decline in food consumption and body weight, emaciation, and occasional diarrhea. There was no change in the concentration of sodium, potassium, calcium, or magnesium in the blood plasma, but the chloride was significantly higher in those given 2.0 per cent NaCl.—*Vet. Bull. (April, 1958): Item 1165.*

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Turkeys 6 months old tolerated concentrations of 6 to 8 per cent of salt in their mash for four weeks. The only effects were smaller gains and a higher water consumption than in turkeys given a 4 per cent salt mash.—*Vet. Bull. (April, 1958): Item 1166.*

Bovine Intoxication from Ingesting Sugar Beets

In October, 1955, 25 dairy cows, in France, that had been fed dry lucerne grass while on pasture were given ground sugar beets with grain and oats instead of the dry grass. The next day, the cows refused the beets, some walked abnormally, and 1 could scarcely rise. Intravenous therapy with glucose, magnesium, and calcium was ineffective. There was no fever and, on necropsy, no inflammatory changes, indicating that death was due to intoxication.

On examination of the stomach and its contents, fluorides, nitrates, nitrites, and toxic metals were absent. Red beets, rich in sugar, were considered the cause of indigestion; yellow-stemmed beets with a much lower sugar content were harmless. A dairy cow could eat 120 lb. of yellow beets daily but not 80 lb. of red beets. However, to feed this quantity, one third of that amount should be fed originally and the quantity increased gradually over a period of two weeks.—*A. Charton et al. in Rec. méd. vét. (Dec., 1958): 1068.*

Vitamin D Intoxication.—A 6-year-old boy who, in six weeks, had been given 3.7 million units of vitamin D, orally and intramuscularly, developed a severe intoxication with all of the typical signs and symptoms. He showed anorexia, nausea, vomiting, thirst, polyurea, polydipsia, constipation, loss of weight, high blood pressure, albuminuria, erythrocyturia, tubal casts, increased erythrocyte sedimentation rate, hypercalcemia, and hypercalciuria.

He was treated with forced fluid intake, low calcium diet, and cortisone. The latter probably prevented a fatal outcome.—*J.Am.M.A. (April 19, 1958): 2089.*

Editorial

Dr. Donald Price Joins Editorial Staff

The Board of Governors is pleased to announce that Dr. Donald A. Price has accepted appointment to the editorial staff of the Association's publications, effective July 1, 1958.

Dr. Donald A. Price

He has been in private practice, in San Angelo, Texas, in partnership with Dr. W. F. Juliff for the past three years. Theirs was chiefly a small animal practice but Dr. Price also did some large animal practice. During the preceding five years, he was engaged in research work on large animal diseases at the Texas Agricultural Experiment Station at Sonora.

Dr. Price was born on Dec. 25, 1919, at Bridgeport, Ohio, where his father operated a creamery. He worked in the creamery when not attending school. He then took a course in business administration and, after graduation, worked in the administration office of the Wheeling Steel Corporation, Wheeling, W. Va.

He entered military service as an infantry private in 1941 and was separated as an Air Force captain in 1946. His military assignments included commandant of cadets, personnel officer, and adjutant.

Dr. Price had always been interested in veterinary medicine, so he enrolled as a postwar student at Ohio State University, Columbus, where he received his D.V.M. degree in 1950. While a student, he was employed during nonclass hours by the Psychology Department as a special vocational counselor. He gave that up during his last two years in veterinary medicine for a laboratory assistantship in veterinary parasitology.

Upon graduation, he was employed as associate veterinarian at the Texas Agricultural Experiment Station, Sonora, where he did investigational work, chiefly on diseases of sheep and cattle. He is best known for his work with bluetongue in sheep which, before the virus was identified, was called "soremuzzle" (JOURNAL, Jan., 1952, page 23).

He also worked with parasitic and breeding problems of sheep and with encephalomyelitis and plant poisoning in cattle. He aided in the production of contagious ecthyma vaccine.

In addition, he made field investigations of diseases of large animals and, at times, functioned as an extension veterinarian in Texas.

He is author or co-author of articles on both large and small animal diseases, including 11 articles in the JOURNAL.

During his military assignment in Texas, Dr. Price met and married Miss June L. Fleming in 1945. They have three daughters: Karen, age 7, Nita, age 5, and Jeane, age 4.

ABSTRACTS

Bacitracin and Growth of Newborn Pigs

Two Hampshire gilts, fed garbage during gestation, and 4 Duroc-Jersey gilts, fed rations meeting nutritive requirements, farrowed 47 pigs. At 36 hours of age and at weekly intervals thereafter, blood samples were taken from the pigs and evaluated. At the time of the first sampling, a pellet containing 1,000 units of bacitracin was implanted subcutaneously in each of 15 pigs, a slow-absorbing zinc-aluminum pellet containing 1,000 units of bacitracin was implanted in each of 16 pigs, and the remaining 16 pigs were left as controls.

The bacitracin treatment did not result in the alteration of the hemoglobin, hematocrit, mean corpuscular volume of erythrocytes, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, number of erythrocytes in the circulating blood, or total leukocytes and nucleated erythrocytes in the circulating blood.

The bacitracin treatment did not result in histological changes in the organs examined.

Growth was not altered as a result of the subcutaneous implantation of bacitracin pellets.

A subclinical anemia was observed in all pigs early in the nursing period.

Pigs from sows fed garbage during gestation had more nucleated erythrocytes in the circulating blood than those fed rations meeting the nutritive requirements.

The presence of nucleated erythrocytes in the blood stream, encountered in certain nutritional deficiencies, appeared to give a higher total leukocyte count than actually existed.—[*Melvin J. Swenson, G. K. L. Underbjerg, Dennis D. Goetsch, and C. E. Aubel: Blood Values and Growth of Newborn Pigs Following Subcutaneous Implantation of Bacitracin Pellets. Am. J. Vet. Res., 19, (July, 1958)* 554-559.]

A Thiry-Vella Fistula in the Chicken

A technique is described for producing a modified Thiry-Vella fistula in the small intestine of the chicken, for use in absorption studies. A loop of intestine, 5 to 7 cm. in length and carrying one and sometimes two radial blood vessels, is resected and the ends exteriorized by means of nylon cannulas. The interrupted intestine is rejoined by a side-to-side anastomosis. Approximately 75 per cent of the 110 prepared specimens functioned satisfactorily. Some birds survived for six months with no apparent ill effects.—[*Howard J. Newman and M. Wight Taylor: A Cannulated Thiry-Vella Fistula in the Chicken. Am. J. Vet. Res., 19, (April, 1958): 473-477.*]

Agglutinative Substances in Anti-Brucella Serums

The titration of *gamma, beta, and alpha* globulin and albumin fractions of Brucella hyperimmune rabbit antiserums was carried out. The titers of *gamma* and *beta* globulin fractions of the serums increased proportionally after a course of immunization.

Ultracentrifugal and electrophoretic analyses were made on rabbit serums obtained before and after a course of immunization and on bovine serums obtained from cattle which were considered "suspicious" when tested for brucellosis. The same results were observed in both analytical methods.

A new attempt, the fixation test following paper electrophoresis, was made on immunized rabbit and bovine serums. The reaction occurred in an area between the *gamma* globulin fraction and the *beta* globulin fraction.—[*Yoshihiro Ozawa and Mitsuru Takanami: Studies on the Agglutinative Substances in Anti-Brucella Serums. Am. J. Vet. Res., 19, (April, 1958): 500-504.*]

Flocculation Tests for Pullorum Disease

Ectoantigens of bacteria possess properties which make them useful in preparing antigens for a flocculation test. This work describes the method of preparing such an antigen emulsion by coating cholesterol with a dialyzed, cell-free *Salmonella pullorum* ectoantigen extract, the procedures for performing the tests, and the standardization of the antigen and lecithin solutions.

The results indicated that the flocculation test will give best reactions when run on heat-activated serum. The positive tests are distinct, and the end point of each dilution is sharp and definite. The flocculation test yields results in a shorter time than the standard tube-agglutination test. The results on 445 serums give an indication of the possible use of such a method as a routine test for pullorum disease in fowl.—[*Edward P. Roznowski, Jr., and V. D. Foltz: Flocculation Tests for Pullorum Disease. Am. J. Vet. Res., 19, (April, 1958): 478-482.*]

Growth of Vesicular Exanthema Virus

Swine kidney cells in tissue culture are capable of supporting the growth of vesicular exanthema virus type B_{51}. This is demonstrated by the fact that, after 20 passages in such a system, representing a dilution from the initial material of 10^{-20}, the virus still manifests typical reactions in swine and in the serum-neutralization test. Titrations of the virus in tissue culture ranged from $10^{6.6}$ to $10^{7.2}$ $i.d._{50}$ per 0.1 ml.

The virus has a cytopathogenic effect on swine kidney cells in tissue culture, which is used as the criterion of viral activity in the serum-neutralization test.—[*Stuart R. Hopkins: The Growth and Cytopathogenicity of Vesicular Exanthema Virus in Swine Kidney Tissue Culture. Am. J. Vet. Res., 19, (April, 1958): 497-499.*]

Treatment of Infectious Sinusitis in Turkeys

Comparisons of the efficacy of several drugs in the treatment of the disease, produced by injecting the left sinus with infective material, indicate that

furazolidone as a 25 per cent aerosol (2 Gm. daily for 5 days) and as a water mix of 4.55 per cent nitrofurazone and 1.77 per cent furazolidone (16 Gm. in 2.5 gal. water for 21 days) is not as effective as drugs injected into the sinus, when observed for 21 days. Ten per cent furazolidone in suspension and in mineral oil, 10 per cent nitrofurantoin in suspension and in mineral oil, streptomycin-dihydrostreptomycin combination (125 mg. of each/ml.), oxytetracycline in mineral oil (25 mg./ml.), and a 4 per cent aqueous solution of silver nitrate were injected in 1-ml. amounts in each sinus after aspiration of the exudate. With the exception of the nitrofurantoin suspension, the streptomycin-dihydrostreptomycin combination in the second experiment, and the silver nitrate solution, there were varying reductions in the sinusitis by the injection of the drugs.—[*W. J. Benton and M. S. Cover: Experimental Treatment of Infections Sinusitis in Turkeys with Nitrofurans and Antibiotics. Am. J. Vet. Res., 19, (April, 1958):* 489-493.]

Pulmonary Adenomatosis in the Chinchilla

Pulmonary adenomatosis was seen in 19 of 206 chinchillas. Affected animals showed signs of dyspnea, anorexia, lethargy, and debility for periods of one to six weeks.

In one flock of chinchillas, the disease reached epizootic proportions, although no etiological agent was isolated. Histologically, the disease consisted of bilateral multicentric foci of interstitial pneumonia containing alveoli lined by nonciliated cuboidal epithelium. These cells were believed to originate from the bronchiolar epithelium. The lesions resembled pulmonary adenomatosis of man and sheep.—[*C. F. Helmboldt, E. L. Jungherr, and A. Cuba Caparo: Pulmonary Adenomatosis in the Chinchilla. Am. J. Vet. Res., 19, (April, 1958):* 270-276.]

Killed Newcastle Disease Vaccines

The results of studies of the comparative immunizing potency of three *beta*-propiolactone-killed Newcastle disease vaccines prepared with the GB Texas, Roakin-NJ, and Manhattan-Kan strains of Newcastle disease virus are presented. A single injection of these vaccines afforded variable degrees of protection to groups of birds vaccinated at 2 weeks and challenged at 4, 6, 8, 10, and 12 weeks of age.

The vaccine made with the GB-Texas strain of Newcastle disease virus afforded the best protection. Groups of birds receiving a second, or booster, injection four weeks after the first injection of vaccine exhibited an anamnestic response that could be measured by the serological and postvaccination virus exposures used in these studies.

When two injections of vaccine were used, no significant difference could be observed in the various groups of birds, irrespective of the strain of virus used in the vaccine.—[*J. F. Sullivan, Eugene Gill, and A. M. Somer: Immune Response of Chickens to Beta-Propiolactone-Killed Newcastle Disease Vaccines. Am. J. Vet. Res., 19, (April, 1958):* 483-488.]

Macroscopic-Slide Test for Leptospirosis

A simple method for the preparation of leptospiral macroscopic-slide agglutination test antigens is described. Different lots of antigens prepared from six leptospiral serotypes were found to be stable for nine to 12 months with little or no variation in sensitivity. Antigens for 12 leptospiral serotypes have been prepared and are used routinely. Comparative tests on 138 human serums, 1,249 bovine serums, 265 canine serums, and 80 porcine serums by slide and microscopic agglutination indicated that the slide test is equally as sensitive as the microscopic test. For rapid screening, the 12 antigens are combined into four pools of three antigens each.—[*Mildred M. Galton, Dorothy K. Powers, Anna D. Hall, and Richard G. Cornell: A Rapid Macroscopic-Slide Screening Test for the Serodiagnosis of Leptospirosis. Am. J. Vet. Res., 19, (April, 1958):* 505-512.]

FOREIGN ABSTRACTS

Adaptation of Aftosa Virus to Rabbits

The authors have been able to transmit any strain of aftosa virus to rabbits less than 8 days of age.

Some changes in virulence were observed in continuous rabbit passage. After 50 rabbit passages, the virulence for cattle remained constant. In guinea pigs, generalized lesions developed in virus cultivated for 20 or more passages.

Vaccination of cattle with fiftieth passage virus produced good protection.—[*J. Verge, A. Paraf, L. Dbennin, Leone Dbennin, and J. Asso: Adaptation of Foot-and-Mouth Virus to Newborn Rabbits. Bull. Off. internat. Epizoot., 49, (1958):* 93-98.]—J. P. SCOTT.

Tissue Culture Aftosa Vaccine

Kidney tissue cultures have been used to prepare aftosa virus having cytogenic titers of $10^{2.75}$ or more per 0.1 cc.

Aluminum hydroxide-swine kidney virus cultures have been prepared and tested in cattle. Field tests have also been successful.

Immunity with tissue vaccine is developed after 20 days. The Waldmann vaccine produced immunity in 14 days.—[*V. Mazzarachio, V. Zavagli, Z. Orfei, A. D'Amore, L. Ravaili, and B. Castagnoli: A Foot-and-Mouth Disease Vaccine Prepared with Tissue Culture Virus Grown on Swine Kidney Tissue. Bull. Off. internat Epizoot., 49, (1958):* 91-92.]—J. P. SCOTT.

Trivalent Aftosa Vaccination

Observations on the recurrence of aftosa in infected herds shows that European herds are menaced by the three fundamental types of aftosa virus.

Vaccination with single or bivalent vaccines have not completely controlled outbreaks.

Since 1952, a trivalent vaccine has been used extensively in Belgium; 5,400,000 cattle have been treated, using 20 cc. of trivalent vaccine.

The trivalent vaccine was tested against each of the three virus types—O, A, and C. None of the vaccinated animals developed generalized lesions, 20 per cent showed no lesions, some others had mild reactions. All unvaccinated controls developed generalized lesions. These results were identical to those found when testing single strain vaccines. Antibody titration against all three types of virus also showed that trivalent vaccines produced as high titers as single type vaccines.

The use of trivalent vaccine may be more expensive than single vaccines, but it produces substantial protection against all three viruses.—[*E. R. Williams and J. Lennin: Bull. Off. internat. Epizoot., 49, (1958): 71-83.*]—J. P. SCOTT.

BOOKS AND REPORTS

Bibliography of Medical Reviews

The third annual volume of this bibliography contains approximately 2,900 references to review articles in clinical and experimental medicine and allied fields, most of which were published in 1957. It is arranged by subject, with a separate author index.—[*Bibliography of Medical Reviews. Superintendent of Documents, U.S. Government Printing office, Washington 25, D. C. 1958. Price (est.) $1.25.*]

Livestock Improvement

The author has done well in closing the gap between the theory and the practice of improving the different breeds of livestock. He writes within the premise that "Formal genetics does not always provide a satisfactory and convenient approach to an understanding of the principles involved in questions of livestock improvement and selection. The breeder finds it too remote at many stages from practical applications and bearings; for the agricultural and veterinary student it usually follows too detached a route. The approach made here represents an attempt to outline the principles and to indicate how the genetic and environmental concepts are interwoven in the idea of livestock improvement."

The text discusses in adequate detail the basic phenomena of the influence of genes upon reproduction. This takes into consideration the hereditary characteristics of the parents, the time and extent of gene influence upon the zygote, embryo, and fetus, and the transmission of characteristics that can be predicted in the offspring. Failures and deformities and death of the fertilized ovum during different phases of gestation, and abnormalities, as in the case of dwarfism, are described and accounted for.

The geneticist will find no fault with the basic approach; the veterinary student will be reminded of the ever-present possibility of genetic causes of infertility; and the breeder will be pleased with the clear and practical handling of improvement problems.—[*Livestock Improvement in Relation to Heredity and Environment. 4th ed. By J. E. Nichols. 240 pages. Oliver and Boyd, Edinburgh: Tweeddale Court, London: 39A Welbeck St., W.1. 1957. Price $3.50.*]—H. E. KINGMAN, Sr.

Pharmacological Principles of Medical Practice

The authors have done a remarkable job in bringing the fourth edition of this book up to date. Although it is a text written primarily for medical students, there are many aspects in this book which make it of value to the veterinary medical profession. It contains 59 chapters which cover aspects of pharmacology. Although the present edition contains the same general arrangement of subject matter as found in the earlier editions, the material has been rewritten, amplified, and revised to present it in line with today's concept of pharmacology.

Two new chapters have been added; one deals with the use of drugs in the treatment of mental illness. This is a discussion dealing with the use of tranquilizing drugs. The other chapter includes discussions on drugs used in the ear, eye, nose, and throat. A more complete discussion of the antihistaminic drugs is included in this edition than in the third edition. There is new matter on antibiotics and other anti-infective drugs in addition to discussions on steroids, hypertensive drugs, and agents used in the control of diabetes.

New concepts of disease and its treatment with drugs acting on the enzyme system have been included. Some older drugs have been deleted from this edition. There is only limited discussion on poisonous drugs and local anti-infective agents. The book is well illustrated and written in a manner so that it is easy to read and understand.—[*The Pharmacologic Principles of Medical Practice. By J. C. Krantz, Jr., and C. Jelleff Carr. 1,313 pages. Illustrated. Williams and Wilkins Co., Baltimore, Md. 1958. Price not given.*]—ROGER LINK.

Diseases and Parasites of Poultry

This is the seventh reprinting of this valuable book since the original in 1935—an indication of its popularity. Its great value to students and research workers lies in the extensive bibliography of 800 references, selected from 8,000 consulted by the authors. The subdivisions of the book are anatomy of the fowl; diseases—bacterial, viral, and protozoan; diagnostic methods; nutritional disorders; parasites—internal and external; and nutrition.—[*Diseases and Parasites of Poultry. By E. H. Barger, L. E. Card, and B. Pomeroy. 5th ed. 408 pages. Lea and Febiger, Philadelphia, Pa. 1958. Price not given.*]—CHARLES MURRAY.

THE NEWS

Programs of Group Conferences in Philadelphia

The following are programs of a few of the meetings to be held in Philadelphia preceding and during the AVMA Convention, Aug. 18-21, 1958.

The Preconvention Conference on "Veterinary Associations and Public Relations" and the Conference of Editors are sponsored by the AVMA.

Preconvention Conference on
Veterinary Associations and Public Relations

Pennsylvania Ballroom, Sheraton Hotel

Sunday, August 17

Chairman—H. E. Kingman, Jr., Director, Interprofessional Relations, AVMA.

9:30 a.m. Coffee and Donuts (Compliments of the AVMA).
10:00 a.m. Strong Local Associations Are the Building Blocks of Public Relations.
W. W. Armistead, President, AVMA.
10:30 a.m. A Strong Local Association In Action.
Bruce Hostrawser, South Bend, Ind.
11:00 a.m. The Women's Auxiliary Role in a Strong Local Association.
Mrs. G. L. Pearce, Royal Oak, Mich.
11:30 a.m. An Example of Effective Local Public Relations—An Emergency Phone Service.
C. E. DeCamp, Scarsdale, N.Y.
Discussion.
12:30 p.m. Luncheon—(Tickets—$2.00)
Local Participation in Science Fairs.
Joseph Kraus, Coordinator, National Science Fairs, Washington, D.C.
2:00 p.m. Organization of an Urban Local Association.
A. G. Madden, Cincinnati, Ohio.
2:30 p.m. Organization of a Rural Local Association.
M. M. Leonard, Asheville, N. Car.
3:00 p.m. Building a Strong State on Local Associations.
W. F. Harris, Puyallup, Wash.
3:30 p.m. Local Veterinary Association Profile (Survey).
H. E. Kingman, Jr.
4:00 p.m. Reflections of PR at National, State, and Local Levels.
B. M. Forster, Director, AVMA Public Information.
Adjournment

The Memorial Chapel at Valley Forge, 20 miles from Philadelphia.

American College of Veterinary Toxicologists

Delaware Valley Suite C, Sheraton Hotel

Sunday, August 17

10:00 a.m. President's Message—*Dr. H. E. Furgeson, Anaconda, Mont.*
10:20 a.m. Problems with Insecticides—*Dr. R. D. Radeleff, Kerrville, Texas.*
10:50 a.m. Progress Report on the Fluorosis Filmstrip—Committee Charting the Course for an Expanded Organization—*Dr. William F. Harris, Secretary, Puyallup, Wash.*
11:20 a.m. Studies in Radiobiology—*Dr. William J. Clarke, Hanford, Wash.*
11:50 a.m. Business Meeting—election of officers—*Dr. H. E. Furgeson, presiding.*
1:00 p.m. Luncheon—Sheraton Hotel.
2:15 p.m. Fluorosis (tentative).
2:45 p.m. Toxicological Aspects of Food Chemistry—*Major Samuel W. Thompson, Denver, Colo.*
3:15 p.m. Selenium Poisoning—*Mr. O. A. Beath, Laramie, Wyo.*

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American Board of Laboratory Animal Medicine

Connie Mack Room, Sheraton Hotel

Sunday, August 17

6:00 p.m. Banquet (fellows and associate members).
7:00 p.m. Report of Secretary-Treasurer (fellows and associate members).
7:30 p.m. Scientific Session (open meeting).
The Care of Dogs for Laboratory Work in Cancer—*E. L. Stubbs and J. F. Shirer, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa.*
Terramycin in the Treatment of Rats in an Outbreak of Infectious Catarrh—*W. C. Doloury, A. L. Hesse, and G. O. MacDonald, College of Medicine, University of Illinois, Chicago, Ill.*
Control of Follicular Acariasis of Mice and Dogs—*R. J. Flynn, Argonne National Laboratory, Lemont, Ill.*
Periarteritis in the Laboratory Rat—*B. H. Skold, Armed Forces Institute of Pathology, Walter Reed Army Medical Center, Washington, D.C.*
9:30 p.m. Meeting of the Council.

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Conference of Editors

Sheraton Hotel—Rooms 556-558

Monday, August 18

Chairman—W. A. Aitken, Editor-in-Chief, AVMA Publications

7:30 p.m. The Technique of Medical Writing.
Walter Kaboe, Director, Medical Department, J. B. Lippencott Company.
Questions and Answers.
8:15 p.m. Testimonial-type of Manuscripts on Therapeutic Agents.
W. R. Riser, Editor, Small Animal Medicine, JOURNAL of the AVMA.
8:30 p.m. Problems in Reproducing Illustrations.
Helen S. Bayless, Assistant Editor in Charge of Production, AVMA Publications.
9:00 p.m. Questions.

Women's Veterinary Medical Association

Delaware Valley Suite A, Sheraton Hotel

Monday, August 18

3:00 p.m. Reception—Officers of the W.V.M.A. (Coffee and cake).
Business Meeting.
Panel discussion—Women in Veterinary Medicine—*Dr. Marge Gullick, Chairman.*
The Veterinarian in Atomic Energy Research—*Dr. Louise Lombard.*
Infertility in Horses—*Dr. Phyllis Lose.*
The Cat and Small Animal Practice—*Dr. Nancy Riser.*
Two Can Practice as Cheaply as One—*Dr. Doris Emmerson.*
The Zoo Veterinarian—*Dr. Patricia O'Connor.*
Discussion.
Presentation of the W.V.M.A. Award.
Movie—The Pakistan National Livestock Show, 1958 (Dr. Harry Steinbach, Officiating Judge).
Dinner—Hotel Dining Room.

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Symposium on Regulatory Veterinary Medicine

Main Lobby, second floor, Convention Hall

Tuesday, August 19

Chairmen—John G. Milligan and Frank A. Todd

1:00 p.m. Film—Hidden Menace.
1:30 p.m. Opening Remarks—*John G. Milligan, Montgomery, Ala., and Frank A. Todd, Washington, D. C.*
1:35 p.m. History of Regulatory Veterinary Medicine—*R. W. Smith, Concord, N. H.*
1:45 p.m. Animal Health and American Agriculture—*Byron T. Shaw, Washington, D. C.*
2:15 p.m. Animal Disease Eradication—Its Benefits to the Producer—*Albert K. Mitchell, Albert, N. M.*

The Administration of Regulatory Programs

Animal Disease Control and Eradication:

State Participation:

2:45 p.m. State Department of Agriculture—*James R. Hay, Columbus, Ohio.*
3:05 p.m. Livestock Sanitary Board—*Ralph L. West, St. Paul, Minn.*
3:15 p.m. Joint State-Federal Program—*Richard W. Carter, Columbia, S. Car.*
3:30 p.m. Federal Participation—*R. J. Anderson, Washington, D. C.*
4:00 p.m. Regulatory Veterinary Medicine in Canada—*Kenneth F. Wells, Ottawa, Ont., Canada.*
4:15 p.m. International Inspection and Quarantine—*L. C. Heemstra, Washington, D. C.*
4:35 p.m. A State's Views on Livestock Import Restrictions and Controls—*Arthur G. Boyd, Sacramento, Calif.*
4:45 p.m. Relationship of Research to Regulatory Veterinary Medicine—*Hadleigh Marsh, Bozeman, Mont.*

Wednesday, August 20

Biological and Pharmaceutical Inspection and Control

8:30 a.m. Biological Products in Interstate and Foreign Commerce—*J. M. Hejl and A. L. Tellejohn, Washington, D. C.*

8:45 a.m. Pharmaceuticals—*John H. Collins, Washington, D. C.*

Food Inspection Programs

Meat Inspection

9:00 a.m. Federal Programs—*A. R. Miller, Washington, D. C.*

9:20 a.m. State Programs—*A. P. Schneider, Boise, Idaho.*

Poultry Inspection

9:30 a.m. Federal Programs—*Roy E. Willie, Washington, D. C.*

9:45 a.m. State Programs—*L. E. Bartelt, Sacramento, Calif.*

Milk Inspection

10:00 a.m. State Programs—*W. L. Bendix, Richmond, Va.*

10:15 a.m. Discussion.

10:30 a.m. Responsibilities of Accredited Practicing Veterinarians—*F. B. Wheeler, Baton Rouge, La.; E. E. Saulmon and T. A. Gage, Washington, D. C.; L. A. Dykstra, Aurora, Ill.*

10:45 a.m. Inter-relationship with Public Health Activities—*Robert D. Courter, Atlanta, Ga.*

Atomic Energy and Regulatory Veterinary Medicine

11:05 a.m. Epidemiological Approaches to Environmental Radiation in Livestock—*Bernard F. Trum, Washington, D. C., and Ted Rea, Fort Worth, Texas.*

11:30 a.m. Peacetime Uses of Atomic Energy.

Screwworm Eradication Program in Southeastern United States —*C. L. Campbell, Tallahassee, Fla., and R. S. Sharman, Washington, D. C.*

Food Preservation—*R. K. Somers and R. H. Philbeck, Washington, D. C.*

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American Society of Veterinary Ophthalmologists

Delaware Valley Suite B, Sheraton Hotel

Tuesday, August 19

Chairman—Dr. W. G. Magrane, Mishawaka, Ind.

7:30 p.m. Ocular Therapeutics—*Dr. Robert Trueman, Graduate School of Medicine, University of Pennsylvania.*

Business Meeting.

Applications Invited for Senior Postdoctoral and Science Faculty Fellowships

Applications will be accepted through Oct. 15, 1958, by the National Science Foundation for both the senior postdoctoral and science faculty fellowships.

Henceforth, these fellowships will be awarded annually in December rather than biannually as awarded in previous years. Awards will be made in the mathematical, physical, and engineering sciences; medical and biological sciences including anthropology and psychology (excluding clinical psychology); and in selected social science fields. Also included are interdisciplinary fields which overlap two or more scientific disciplines. The names of successful fellowship candidates will be announced on Dec. 5, 1958.

To be eligible for senior postdoctoral fellowships, candidates must be citizens of the United States with special aptitude for advanced training and must have held the doctoral degree for at least five years or have the equivalent in education and experience. Senior postdoctoral fellows will be selected on the basis of ability as evidenced by letters of recommendation and by personal achievement.

The science faculty fellowships are directed toward college teachers of science who wish to improve their competence as teachers. These fellowships are open to application by any citizen of the United States, who holds a baccalaureate degree or its equivalent, has demonstrated ability and special aptitude for science teaching and advanced training, has taught at the collegiate level as a full-time faculty member for not less than three years, and intends to continue teaching. Selection will be based on letters of recommendation, academic records, and other appropriate evidences of professional competence.

Annual stipends up to $12,000, adjusted to match as closely as possible the regular salaries of the recipients, will be awarded under both these programs. Fellows may engage in study and research at any accredited nonprofit institution of higher learning in the United States or any similar institution abroad approved by the National Science Foundation. A limited allowance to aid in defraying the cost of travel for a fellow and his dependents will also be available.

Application materials may be obtained from the Division of Scientific Personnel and Education, National Science Foundation, Washington 25, D. C.

Symposium on Regulatory Veterinary Medicine in Philadelphia

The annual work conference for state and federal regulatory veterinarians to be held during the AVMA convention in Philadelphia is scheduled for Tuesday, August 19, at 1:00 p.m. on the second floor of the Main Lobby of Convention Hall (*see* p. 136 for program).

The symposium is under the cosponsorship of the U.S. Livestock Sanitary Association and the Agricultural Research Service, in cooperation with the AVMA.

The members of the committee in charge of arranging the program are: Dr. Frank A. Todd, chairman, and Dr. John G. Milligan, cochairman; Drs. C. L. Campbell, R. W. Smith, A. P. Schneider, A. R. Miller, and C. D. Van Houweling.

Dr. Morter Receives National Science Foundation Fellowship

Dr. Raymond L. Morter (ISC '57), currently engaged in graduate work in the College of Veterinary Medicine at Michigan State University, was appointed a National Science Foundation regular postdoctoral fellow on March 15, 1957.

Dr. Raymond L. Morter

Dr. Morter is one of the first veterinarians to obtain this honor. The research program for his doctoral dissertation is being conducted under the guidance of Dr. E. V. Morse in the field of bovine leptospirosis.

At Michigan State University, Dr. Morter is majoring in microbiology with minors in veterinary pathology and physiology.

Dr. R. S. Sharman Wins Public Administration Award

Dr. Robert S. Sharman (API '45), Arlington, Va., U. S. Department of Agriculture veterinarian and former co-director of the Mexican-United States Commission for Prevention of Foot-and-Mouth Disease, was presented the William A. Jump Memorial Award in special ceremonies in Washington, May 27, 1958.

The award is presented annually in recognition of outstanding leadership and exemplary achievement in the field of public administration. James M. Mitchell, chairman of the Jump Memorial Foundation board of trustees, made the presentation.

Dr. Sharman joined the U.S.D.A. as a livestock inspector. He was a member of the initial group of veterinarians sent to Mexico in 1947 to help fight foot-and-mouth disease there. In 1955, he became co-director of the commission.

Recently, Dr. Sharman has been named to head the U.S.D.A.'s portion of the federal-state campaign to eradicate the screwworm from the southeastern United States.

s/T. F. McGINTY, *U.S.D.A., Information Division.*

Dr. Steele Receives Alumni Award from MSU

Dr. James H. Steele, chief veterinarian of the U. S. Public Health Service's Communicable Disease Center, Atlanta, received the Alumni Award for Distinguished Service from Michigan State University at commencement exercises in East Lansing on June 8, 1958.

Dr. Steele is one of four who received the honor this year. The award is made annually by the Michigan State University Alumni Club of Washington, D. C., in cooperation with the university.

Dr. Steele is author of many articles on veterinary public health and lecturer at various universities, including Harvard, Yale, and Minnesota.

AMONG THE STATES AND PROVINCES

Alberta

Alberta V.M.A. Meet in Banff.—The Alberta Veterinary Medical Association held their annual meeting in beautiful Banff on June 20-21, 1958.

Among the speakers were Dr. H. C. Smith, Allied Laboratories, Sioux City, Iowa, who spoke on swine diseases; Dr. George E. Short, Eaton Laboratories, Norwich, N.Y., discussed infectious diseases of swine; and Dr. Damon V. Catron (Ph.D.) of Iowa State College, Ames, spoke on nutrition in swine.

The Honorable Douglas S. Harkness, Minister of Agriculture and member of Parliament for Calgary North, was the guest speaker at the banquet held at the Timberline Hotel on June 20.

California

Dr. K. F. Meyer Given Honorary LL.D Degree.—Dr. Karl F. Meyer, former and now emeritus professor of research medicine and director of the Hooper Foundation for Medical Research of the University of California, was given the honorary degree of Doctor of Laws by the University in a ceremony held last March. This honor was in recognition of his contributions to public health through his scientific and social awareness and effectiveness.

Dr. Meyer received the D.V.M. degree in 1909 from the University of Zurich, Switzerland, came to the United States in 1910, was professor of pathology and bacteriology at the School of Veterinary Medicine, University of Pennsylvania, from 1910 to 1913, then became professor of bacteriology and protozoology at the University of California. In 1915, he became associated with the Hooper Foundation.

In addition to the D.V.M. degree, Dr. Meyer received his Ph.D. from Zurich in 1924; M.D., College of Medical Evangelists in 1937; Dr. Med.h.c., Zurich, in 1937; LL.D., University of Southern California, 1946, and is the recipient of many other honors.

Iowa

Death Comes to Mrs. Q. D. Sundberg.—Mrs. Quenten D. Sundberg, Linn Grove, wife of Dr. Sundberg (ISC '55) and the daughter-in-law of Iowa's state veterinarian, Dr. A. Lawrence Sundberg (ISC '31), died in a Sioux City hospital after a ten-day illness believed to have been encephalitis.

Surviving Mrs. Sundberg, in addition to her husband, are a daughter and two sons.

Kansas

KSC's Veterinary Science Hall Named in Honor of Dr. Burt.—The veterinary science hall at Kansas State College was named in honor of Dr. James H. Burt (ONT '95), emeritus professor of anatomy, who served on the veterinary staff for 38 years.

The dedication took place in a brief ceremony June 6, 1958, during the twentieth annual conference of Kansas veterinarians in Manhattan.

The veterinary science hall at Kansas State College, recently named Burt Hall in honor of Dr. J. H. Burt. The building is now being remodeled.

Dr. Burt joined the college's staff in 1909 and later served as head of the department of anatomy and physiology. When two departments were formed, Dr. Burt became head of the anatomy department. He retired in 1947.

The veterinary science hall is currently being remodeled at a cost of $150,000 to permanently house the departments of pathology and physiology. Some animal disease investigations also will be conducted in the building.

Manitoba

Women's Auxiliary.—The annual meeting of the Women's Auxiliary to the Manitoba V.M.A. was held in the new Carriage Room of the Town 'n Country Restaurant, Winnipeg. Fifty-two members were present from Greater Winnipeg and the rural areas. The Auxiliary has increased to 70 members with 98 per cent of the women also belonging to the Women's Auxiliary to the Canadian V.M.A.

This year the Manitoba Auxiliary has contributed to the C.V.M.A. Loan Fund, the AVMA Research Fund, and the AVMA Memorial Fund. The donation to the Memorial Fund was made in memory of two of the auxiliary's charter members: Mrs. W. Hilton, the auxiliary's first president; and Mrs. W. Spiers.

In an attempt to enlighten the younger generation on the importance of the veterinary profession, speakers have been sent to address high school seniors in a program sponsored by the auxiliary for the first time this year. Other talks by leading veterinarians are being planned.

The auxiliary's officers for 1958 are as follows: Mrs. J. R. English, president; Mrs. N. H. Peter, first vice-president; Mrs. D. N. English, second vice-president; Mrs. D. J. Lawson, third vice-president; Mrs. J. R. Singleton, secretary; and Mrs. Z. Jablonski, treasurer.

s/MRS. BERYL C. SINGLETON, *Secretary.*

Michigan

Dr. Armistead Appointed to Science Advisory Board.—Veterinary medicine and the veterinary profession were given recognition last April when Gov. G. Mennen Williams appointed Dr. W. W. Armistead of the College of Veterinary Medicine, Michigan State University, to a newly created Science Advisory Board composed of representatives of industry and education.

The Board has been asked to study and make recommendations as to the means Michigan can use to better coordinate its existing scientific facilities, insure that its scientific resources are properly integrated into national defense, improve its educational institutions at all levels to guarantee a constant flow of trained scientists and engineers, as well as to help make careers in science more attractive to young people.

Chairman of the 17-man board is Dr. Russell Bright, associate dean and former chairman of the chemistry department, Wayne State University.

Missouri

Greater St. Louis Association.—The following officers were installed by the Greater St. Louis V.M.A. at their meeting on June 6, 1958: Drs. C. M. Ruck, East St. Louis, Ill., president; C. R. Plegge, St. Louis, vice-president; E. E. Epstein, St. Louis, secretary; and J. R. Seelbach, University City, treasurer.

s/E. E. EPSTEIN, *Secretary.*

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Women's Auxiliary.—The Women's Auxiliary to the Greater St. Louis V.M.A. held its annual installation of officers at the home of Mrs. George Murphy, Kirkwood, on May 20, 1958. Mrs. Anthony A. Bott presided.

The following officers were inaugurated: Mrs. C. Herman Beckman, St. Louis, president; Mrs. A. Elmer Blum, Crystal City, vice-president; Mrs. Ernest S. Hernady, St. Louis, secretary; Mrs. James R. Seelbach, University City, treasurer; and Mrs. Richard A. Binder, Lemay, historian.

s/MRS. ALLEN B. SHOPMAKER, *Public Relations Chairman.*

New Jersey

Newly Appointed Examining Board Announced.—The newly appointed State Board of Veterinary Medical Examiners comprises the following officers and members: Drs. J. A. S. Millar, Deal, president; John S. Gray, Newton, vice-president; Robert R. Shomer, Teaneck, secretary-treasurer; and Arthur F. North, Jr., Somerville, and Irving Botwinick, Margate, members.

s/ROBERT R. SHOMER, *secretary-treasurer.*

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Dr. Ives at 85 Retires from Second Career.—Dr. Leland D. Ives (AVC '96), Cranford, who left retirement ten years ago to take over the annual rabies control program in Elizabeth, has declined a board of health request to conduct the rabies program again this year.

Dr. Ives now plans to devote himself to his grandchildren, his vegetable garden, and reading. He had formerly served with the U.S. Bureau of Agricultural Marketing Service as a marketing specialist.

New York

New York City Association.—The regular meeting of the Veterinary Medical Association of New York City, Inc., was held at the New York Academy of Sciences on June 4, 1958.

Dr. Robert W. Kirk, professor, Department of Therapeutics and Small Animal Diseases, New York State Veterinary College, Cornell University, Ithaca, discussed the infectious diseases of cats.

On June 18, the association showed a group of five medical films which included arteriosclerosis, cardiac arrest, acute abdominal injuries, use of the artificial kidney, and the abdominal hysterectomy.

New York State Veterinary College Honor Day Exercises.—The annual Honor Day exercises of New York State Veterinary College were held May 20, 1958, in Willard Straight Hall. The following awards were presented:

The Borden Veterinary Scholarship Award ($300) to the member of the fourth year class who attained the highest scholastic record in all veterinary studies for the first three years: Alexander de Lahunta.

The Horace K. White Prizes for the highest academic records for the entire veterinary course: Alexander de Lahunta, first ($75); and Erwin G. Pearson, second ($25).

The Grant Sherman Hopkins Prize ($30) for interest, ability, perseverance, and performance in the Department of Anatomy: Erwin G. Pearson.

The Jane Miller Prizes to members of the second year class for the best work in veterinary physiology: Richard O. Davies, first ($25); Robert D. Phemister, second ($15); and Tobias Jungreis and Anthony D. Caciagli, honorable mention.

The James Gordon Bennett Prizes for the students who show the greatest humaneness in handling animals, with special reference to the use of anesthesia: Louis V. Bowers, first ($25); and Alexander de Lahunta, second ($15).

The Anne Besse Prizes for the best work in medicine and clinical diagnosis: Donald E. Mielke and Harry S. Russell ($20 equally); Alexander de Lahunta and Erwin G. Pearson, honorable mention.

The Charles Gross Bondy Prizes for the best work in the courses in practical medicine and surgery of the small animals: Andre Lavignette, first ($25); Alexander de Lahunta, second ($15); and Louis V. Bowers, honorable mention.

The Mary Louise Moore Prize ($40) for the best work in bacteriology: Alexander de Lahunta.

The Alpha Psi Prize for the student best equipped to advance standards of veterinary science ($25 U.S. Saving Bond): Donald Q. Eno.

The Sigma Iota Zeta Prize for a sincere effort of high purpose and achievement toward his profession ($25 U.S. Saving Bond): Leonard Wood.

The New York State Veterinary Medical Society Prizes for the best case reports presented for publication in *Veterinary News*: Richard E. Hoffer, first ($25); Arnold Remson, second ($15); and Charles Hjerpe, third ($10).

The Prize of the Women's Auxiliary to the AVMA for special contributions of an extracurricular nature ($50): Donald O. Wirth.

The Jacob Traum Award for superior interest and accomplishment in bacteriology, epizootiology, pathology, and virology, with an expressed interest in research on infectious diseases: Erwin G. Pearson.

Quebec

Dr. Filion Speaks at Delaware Conference.—Dr. Roland Filion, connected with the Laboratory of Veterinary Research and professor of veterinary medicine at the University of Montreal, St. Hyacinthe, was one of nine speakers to participate in a symposium on nitrofuraans in Wilmington, Del., June 4, 1958.

Dr. Filion discussed the activity of Furacin on the staphylococci isolated from the mammary gland.

s/JACQUES SAINT-GEORGES, *Secretary.*

Virginia

Two-Way Seminar.—On May 15, 1958, the Chesapeake and Potomac Telephone Company installed a two-way communication system in the home of Dr. John Wittig, Portsmouth, to enable the members of the Tidewater V.M.A. to hear an address by Dr. G. F. Otto, Ph.D., of Abbott Laboratories, Chicago.

Following the discussion, 15 members of the association participated in an hour-long question-and-answer period with Dr. Otto. He discussed heartworms in dogs, emphasizing that instead of a series of injections over a period of two or three weeks which had been required, research in the disease has now made it possible to successfully treat this condition in three to four days.

The Tidewater V.M.A. plans to use this two-way communication again in the near future, as it insures the association informative and interesting programs that would not otherwise be possible.

s/W. R. WEST, *Correspondent.*

U.S. GOVERNMENT

Veterinary Personnel Changes.—The following changes in the force of veterinarians in the U.S.D.A. are reported as of April 29-30 and May 5-28, 1958.

TRANSFERS

Joseph L. Blair, from Salt Lake City, Utah, to Fort Worth, Texas.

Charles D. Chase, from Worthington, Minn., to Central City, Neb.

Jason W. Copeland, from Athens, Ga., to Athens, Ala.

Marvin L. Crutcher, Jr., from Marshall, Mo., to Chicago, Ill.

William Downey, from Denver, Colo., to Montpelier, Vt.

Roger C. Feldman, from Atlantic, Iowa, to Altura, Minn.

John Halij, from Concord, N. Car., to Brooklyn, Conn.

James D. Hatton, from Evansville, Ind., to New York, N. Y.

Wayne A. Hornaday, from Lakewood, N. J., to Philadelphia, Pa.

Mahlon B. Huffman, from Frankfort, Ky., to Baton Rouge, La.

Arthur J. Kowalk, from Cheyenne, Wyo., to Baltimore, Md.

Roy A. Lantz, from Atascadero, Calif., to Seattle, Wash.

Everett O. Lummis, from San Antonio, Texas, to Lake Charles, La.

James M. Mathis, from Houston, Texas, to Kansas City, Kan.

Harry Oja, from Modesto, Calif., to Salem, Ore.
Bill Parker, from Lake Charles, La., to Houston, Texas.
Charles F. Partridge, from Boaz, Ala., to Athens, Ga.
W. L. Pinckard, from Baltimore, Md., to Richmond, Va.
Nicola Romano, from Chicago, Ill., to Los Angeles, Calif.
Forrest A. Underwood, from Frankfort, Ohio, to Chicago, Ill.
Donald A. Yandell, from Salem, Ore., to Turlock, Calif.

RETIREMENTS

Thomas H. Applewhite, Jacksonville, Fla.
Vernon B. Hill, Phoenix, Ariz.
Lynne F. Holmes, Cheyenne, Wyo.
Melvin W. Miller, Hanford, Calif.
Ralph S. Mourning, Kansas City, Kan.
Earl M. Rundahl, Omaha, Neb.
Alonzo V. Wardall, Waterloo, Iowa.

STATE BOARD EXAMINATIONS

Interested persons can obtain information about applications, fees, deadlines for filing applications, and exact time and place of examinations of the respective boards by writing to the persons whose names and addresses are given below.

BRITISH COLUMBIA—November, 1958 (usually end of the second week); Vancouver. G. L. Stovell, British Columbia Veterinary Association, 3187 West 43rd Ave., Vancouver 13, secretary.

KENTUCKY—July 28, 1958. University of Kentucky, Lexington. J. K. Bushnell, secretary, 229 Houston, Paris, Ky.

COMMENCEMENTS

Graduating Class, 1958, School of Veterinary Medicine, Alabama Polytechnic Institute

Front row (left to right)—Walter White Williams, John Duncan Powell, Robert Louis Nelson, Charles Everett Short, Dr. F. E. Hull (Kentucky), Dr. N. D. Peacock (Tennessee), Dr. J. W. Scales (Mississippi), Dean M. A. Brooker (Florida).

Second row—Harry Clay Current, Fred Kenneth Soifer, Gerald Dale Lee, Billie Luella Adels, Edward Sinclair Ford, Jr., Stuart Pugh Dowling, Scott Thomas Green.

Third row—Phillip Hayes Hamm, John Parker Pogue, Hugo Oscar Hempel, III, Donald Butler Popejoy, Robert Oglesby Bickham, Robert Ray Kuykendall, Henry Duke Watson, IV.

Fourth row—David Carl Musselman, Jr., Floyd Russell Crum, John Lincoln Porter, Herschel Wayne Cotton, David Harry Kelly, Billy Olen Blackburn.

Fifth row—William Robert Klemm, James Carroll Woodard, Henry Tyler Fairleigh, John Euclid Gray, James Arthur France, Wiley Devine Hales.

Sixth row—William Oliver Cowart, J. C. Rudd, William Wilford Dugger, Jr., James Howard Poole, Henry Watson Hall, Jr., Vernon Lee Carter, Jr.

Seventh row—James Thomas Wooldridge, George Edward Dyer, Willie Joe Ray, Jr., Homer Ray Meads, Roy Johanson Brinkman, Melvin David Stein.

Eighth row—Jimmy Ray Peacock, Frank Gray Handley, Charles Nunley Murphy, Ewell Powell Conyers, Samuel Wayne Keyes, Gene Austin Bingham.

Ninth row—James Benton Pardue, Robert Lanier Rollins, Jr., James Cameron Lawrence, Wilfred Lorie Collette, John Thomas Sanders, Donald Sherwood Brown, Thomas Lynwood Barber, Donald Dean Bryan, Marshall Hatcher Bryan, Marcus Ferrel Martin, Max James Freeman, Gaylard Parker Willard, Glenn Stuart Boatright, Jr., William Lamar Molpus, John Lamar Anthony, Jr.

Alabama Polytechnic Institute.—At the 1958 commencement exercises of the School of Veterinary Medicine, Alabama Polytechnic Institute, the following 63 candidates were presented for the D.V.M. degree:

Billie Luella Adels
John Lamar Anthony, Jr.
Thomas Lynwood Barber
Robert Oglesby Bickham
Gene Austin Bingham
Billy Olen Blackburn
Glenn Stuart Boatright, Jr.
Ray Johansen Brinkman
Donald Sherwood Brown
Donald Dean Bryan
Marshall Hatcher Bryan
Vernon Lee Carter, Jr.
Wilfred Lorie Collette
Ewell Powell Conyers
Herschel Wayne Cotton
William Oliver Cowart
Floyd Russell Crum
Harry Clay Current
Stuart Pugh Dowling
William Wilford Dugger, Jr.
George Edward Dyer
Henry Tyler Fairleigh
Edward Sinclair Ford, Jr.
James Arthur France
Max James Freeman
John Euclid Gray
Scott Thomas Green
Wiley Devine Hales
Henry Watson Hall, Jr.
Phillip Hayes Hamm
Frank Gray Handley
Hugo Oscar Hempel, III
David Harry Kelly
Samuel Wayne Keyes
William Robert Klemm
Robert Ray Kuykendall
James Cameron Lawrence
Gerald Dale Lee
Marcus Ferrel Martin
Homer Ray Meads
William Lamar Molpus
Charles Nunley Murphy
David Carl Musselman, Jr.
Robert Louis Nelson
James Benton Pardue
Jimmy Ray Peacock
John Parker Pogue
James Howard Poole
Donald Butler Popejoy
John Lincoln Porter
John Duncan Powell
Willie Joe Ray, Jr.
Robert Lanier Rollins
J. C. Rudd
John Thomas Sanders
Charles Everett Short
Fred Kenneth Soifer
Melvin David Stein
Henry Duke Watson, IV
Gaylard Parker Willard
Walter White Williams
James Carroll Woodard
James Thomas Wooldridge

• • •

Graduating Class, 1958, New York State Veterinary College, Cornell University

Cornell University.—At the 1958 commencement exercises of the School of Veterinary Medicine, Cornell University, the following 50 candidates were presented for the D.V.M. degree:

Robert Altman
Efstratios Apostolides
Eric G. Archer
Harold H. Bond, Jr.
Louis V. L. Bowers
James B. Brayton
Berkeley D. Briggs
Ronald L. Buchanan
Robert L. Burgess
Clarence M. Burgher
Miller A. Cook
Raymond W. Cotter
Alan E. Crawford
Richard L. Crissey
Alexander de Lahunta
Beatrice S. English
Donald Q. Eno
John F. Ferrell
Elverd J. Frink
Mark J. Geronimus
Jock Allen Gorelick
Clinton M. Greenwood
B. Sheldon Hagar, Jr.
Charles A. Hjerpe
Theodore Hoch
Richard E. Hoffer
Dorothy Holmes
Wallace Holmes
Samuel Hutchins III
John L. Jackson
George Jordan
Waltraut M. Koeth
Eugene M. Kravis
Andre M. Lavignette
Donald E. Mielke
William E. Moore

Mark L. Morris, Jr.
James T. Noonan
Thomas F. Nytch
Frederick W. Oehme
Erwin G. Pearson
Earle N. Peterson
John E. Post
Edwin L. Rague
Robert G. Rasey
Arnold Remson
Harry S. Russell
Myron G. Schultz
Donald O. Wirth
Leonard Wood

• • •

University of Missouri.—At the 1958 commencement exercises of the School of Veterinary Medicine, University of Missouri, the following 29 candidates were presented for the D.V.M. degree:

James E. Baker
Richard S. Barnett
Robert J. Basnett
Louis M. Benson
James E. Breazile
Kenneth H. Buchmann
Ronald L. DeHaven
William C. Dierberg
Glenn A. Fairchild
John J. Frank
Ward A. Goss
Allen W. Hahn
Harold E. Hayden
Joel F. Hooker
William H. Hughes
Alanson L. Jones
Emil F. Kuhn
William T. London
Charles E. Martin
Gerald L. McEuen
Charles C. Middleton
Lester D. Nordyke
Thomas W. Overhulse
Eugene W. Plegge
Homer E. Steinhoff
Ruth S. Thomure
Jack Lee Vaughn
Charles J. Vosbrink
Billy G. Wright

• • •

Ontario Veterinary College.—At the 1958 commencement exercises of the School of Veterinary Medicine, Ontario Veterinary College, the following 52 candidates were presented for the D.V.M. degree:

Edward Norman Asselstine
Harold Gustav Baer
Frederick Bartolf
Sally Toutant Beauregard
Peter Biliski
Carole Ann Billin
Robert Barmore Billin
James Kenneth Bodendistel
Frances Hazel Calverley
John Leveson Calverley
Roderick Reid Davies
Leigh Bertram Donald
Kenneth Lewis Easton
Julia Eros
Ronald Colling Finlay
Lloyd Daniel Fisher
Donald Fishman
James Michael Ford
Barbara Joan Forsyth
John Allen Forsyth
Donald Patterson Gray
Lorne Everett Greenaway
Harvey Harold Grenn
George Jack Heath
Hugh James Hill
Donald Keith Hills
Harold Roy Johnston
Christian John Lantink
Rudolph George Leibold
Keith Elroy Marling
Douglas Smith Miller

• • •

Graduating Class, 1958, School of Veterinary Medicine, University of Missouri

Top row (left to right)—James E. Baker, Richard S. Barnett, Robert J. Basnett, Louis M. Benson, James E. Breazile, Kenneth H. Buchmann, Ronald L. DeHaven.

Second row—William C. Dierberg, Glenn A. Fairchild, John J. Frank, Ward A. Goss.

Third row—Allen W. Hahn, Harold E. Hayden, Joel F. Hooker, William H. Hughes.

Fourth row—Alanson L. Jones, Emil F. Kuhn, William T. London, Charles E. Martin, Gerald L. McEuen, Charles C. Middleton, Lester D. Nordyke.

Fifth row—Thomas W. Overhulse, Eugene W. Plegge, Homer E. Steinhoff, Ruth S. Thomure, Jack Lee Vaughn, Charles J. Vosbrink, Billy G. Wright.

Graduating Class, 1958, Ontario Veterinary College, University of Toronto

Richard Bell Miller
James Alexander McDonald
Kenneth Alexander McKay
Donal Bryce McKeown
Ian Patrick McMorland
Arthur Terence O'Connor
Paul Wilton Pennock
Victor Ivan Reed
Charles Frederick Rhoads
John Allan Roberts
William David Schaefer
Meryl Percy Schooley
David Cartwright Secord
Duncan Gordon Sinclair
Harry John Smith
Russell Irwin Thompson
Ellen Fleming Thomson
Albert Cornelis van der Meulen
Michael John Walcroft
Clifford Ronald Widdup
John Sheldon Zavitz

• • •

Washington State College.—At the 1958 commencement exercises of the College of Veterinary Medicine, State College of Washington, the following 48 candidates were presented for the D.V.M. degree:

Lee Bennett
Clayton Burnum
Jack Carkeek
George Clarke
Donald Crimmins
Robert Dolphin
Conrad Donovan
John Duff
Ford Ebner
Ervin Erickson
Charles Gardner
Gerald Gardner
Neil Haley
Richard Hall
Garner Harston
Donald Jenkins
Jack Jelmberg
Dean Jensen
Larry P. Jones
George Klavano
Lloyd Lauerman
John Lee
Raymond Loan
Richard Long
Robert Lunger
Jody C. Marshall
Wes Marshall
Warren Mc Culloch
Dean Miller
Robert Mohr
William Noble
David Olney
Glen Overturf
Theodore Peterson
Max Perry
Douglas Philips
Vernon Reitan
Allen Schauffler
Frank Shackelford
William Keith Smith
Fred Stump
DeVon Terry
Jack K. Ward
James Ward
Eugene Wegner
James Williams
Clyde Whiteaker
Roy P. Wright

(Washington State College commencement picture on following page)

Graduating Class, 1958, College of Veterinary Medicine, State College of Washington

—Hutchison, Pullman.

Top row (left to right)—Lee Bennett, Clayton Burnum, Jack Carkeek, George Clarke, Donald Crimmins, Robert Dolphin, Conrad Donovan, John Duff.

Second row—Ford Ebner, Ervin Erickson, Charles Gardner, Gerald Gardner, Neil Haley, Richard Hall, Garner Harston, Donald Jenkins.

Third row—Jack Jelmberg, Dean Jensen, Larry P. Jones, George Klavano, Lloyd Lauerman, John Lee, Raymond Loan, Richard Long.

Fourth row—Robert Lunger, Jody C. Marshall, Wes Marshall, Warren McCulloch, Dean Miller, Robert Mohr, William Noble, David Olney.

Fifth row—Glen Overturf, Theodore Peterson, Max Perry, Douglas Phillips, Vernon Reitan, Allen Schauffler, Frank Shackelford, William Keith Smith.

Sixth row—Fred Stump, DeVon Terry, Jack K. Ward, James Ward, Eugene Wegner, James Williams, Clyde Whiteaker, Roy P. Wright.

DEATHS

Star indicates member of AVMA

★**John E. Bender** (OSU '13), 79, Nashville, Tenn., a pioneer in the control of animal diseases, died in his home following a heart attack.

Dr. Bender had lived in Nashville for the past 32 years and had worked for the Bureau of Animal Industry, U.S.D.A., for 44 years. He was one of the first veterinarians in the country to begin a systematic program for the eradication of disease-bearing ticks in the South. After Dr. Bender retired from his work with the U.S.D.A. in 1949, he became meat inspector for Davidson County and the city of Nashville.

Surviving are a son, Dr. John Bender (OSU '35), Columbus, Ohio, and a brother in North Canton, Ohio.

C. F. Bontrager (OSU '07), 76, Fort Recovery, Ohio, died in his home May 6, 1958, after an illness of several weeks.

For many years, Dr. Bontrager had been veterinary inspector for Swift & Company in Cleveland.

He is survived by his widow and two daughters.

★**Col. E. J. Cramer** (AVC '92), 86, V.C., U.S. Army (retired), San Diego, Calif., died March 22, 1958, in San Diego.

Colonel Cramer was born in Alsace Lorraine in 1872. He became a major in the Veterinary Corps in June, 1920, and a lieutenant colonel in January, 1921. He retired while serving in El Paso, Texas, at his own request after 30 years of service.

He had lived in San Diego since his retirement. Colonel Cramer became a life member of the AVMA in 1951.

★**William C. Edwards** (GR '06), 74, Cambria, Wis., died in a Portage hospital after a brief illness.

Dr. Edwards was a life-long resident of Cambria and had been a veterinarian there for more than 50 years. He was active in the Farmers Union activities. He is survived by his widow and two daughters.

★**Joseph H. Evans** (MCK '17), 66, Genesee Depot, Wis., died at his home May 5, 1958.

Dr. Evans was born in the town of Genesee and was a veteran of World War I. He belonged to the Waukesha Masonic Lodge, and the Dousman Order of Eastern Star.

He is survived by his wife, two daughters, and one son.

★**Leonard R. Haubrich, Sr.** (UP '11), 71, Claremont, N.H., died suddenly at the Claremont General Hospital on April 29, 1958. He had undergone an emergency appendectomy on April 28.

Educated in the local schools in Claremont, Dr. Haubrich practiced in Bernardsville, N.J., after graduation from the University of Pennsylvania, from 1912 to 1919. A few years later he founded the Windyknoll Kennels.

Dr. Haubrich was milk and meat inspector in Claremont from 1920 to 1956, one-time health officer, a member of the Claremont A.S.P.C.S., a federal veterinarian in animal disease work until a few years ago, and for some time assistant state veterinarian, the first in N.H. to ever conduct herd inspection in the eradication of bovine tuberculosis. In February, 1956, Dr. Haubrich received a Rotary certificate of recognition for outstanding service and accomplishment to his community.

He is survived by his wife, Helen Page Haubrich; two sons: one a veterinarian, Wilson R. (UP '37), and Dr. Leonard R., Jr.; and a daughter.

★**Harold E. Johnston** (ISC '13), 67, Readlyn, Iowa, died in St. Francis Hospital in Waterloo, Iowa, on April 24, 1958.

Dr. Johnston was born in Keosauqua, Iowa, in 1890, the son of Francis and Louise Stewart Johnson. He graduated from Keosauqua High School and then went on for his D.V.M. degree at Iowa State College. Dr. Johnston became a life member of the AVMA in 1956.

He is survived by his widow, Mrs. Harold Brownlee Johnston, and three daughters.

★**W. E. Jones** (CIN '15), 68, Leesburg, Ohio, died at Fayette Memorial Hospital following an illness of several months.

Dr. Jones had practiced in Leesburg for 42 years. He was a World War I veteran and a member of the Ohio State V.M.A.

He is survived by his widow, two sons, and a brother.

Ivan L. Hawkins (ISC '18), 66, Chicago, Ill., died suddenly on Jan. 2, 1958.

Dr. Hawkins was the youngest son of Benjamin and Mary (Hill) Hawkins, pioneers of Cascade, Iowa. He served in the Veterinary Corps during World War I, and following his military duty, returned to Cascade and entered veterinary practice and farming with his brother, Arthur. Ten years ago he retired and moved to Chicago. He was subsequently married to Virginia Wray on Aug. 15, 1950.

Dr. Hawkins is survived by his wife, Mrs. Ivan Wray Hawkins, Chicago; four brothers, and two sisters.

John F. Kane (UP '11), 68, Goshen, N.Y., died in Goshen on Jan. 27, 1958.

Dr. Kane owned and operated the Orange County Veterinary Hospital in Goshen for many years. He served in World War I, first in the Italian Army, transferring later to the British, and then to the U.S. Army, where he

was commissioned a second lieutenant in the Veterinary Corps.

He is survived by his widow and a daughter.

★**A. T. Knowles** (KCV '04), 82, one of the leading veterinarians of South Florida and the father of Drs. Jack O. and Robert P. Knowles, Miami, Fla., died on April 22, 1958.

Born at Appleton City, Mo., in 1876, Dr. Knowles practiced in Montana and also served as a federal inspector there for a number of years following graduation from Kansas City Veterinary College before locating in Miami. He was one of the first to report the incidence of heartworms in dogs and, with his sons with whom he practiced, co-authored many published articles on canine medicine.

He was a member of the AVMA, the Florida V.M.A., a life member of the South Florida Veterinary Society, and of the American Animal Hospital Association. He also belonged to various Masonic bodies, the Shrine, the Miami Optimist Club, and was a member of the board of the White Temple Methodist Church.

Surviving besides Dr. Knowles' two sons are his wife; another son, Taylor S.; and two daughters, a brother, and nine grandchildren.

Thomas A. McAllister (ONT '08), 82, Kinde, Mich., died in Hubbard Memorial Hospital, after an illness of three weeks.

Dr. McAllister has resided in Kinde since 1907. He is survived by his wife, Alice Sparling McAllister, two daughters, and two sons.

A. A. Mill (IND '14), 74, Rushville, Ind., died at the Rushville Memorial Hospital on April 12, 1958.

Dr. Mill had been ill for the past three years. He had retired from practice seven years ago. He is survived by his widow, Mrs. A. Edwards Mill.

Percival B. Silvester (MCK '16), 71, Princeton, N.J., died April 21, 1958.

Dr. Silvester is a former member of the New Jersey V.M.A. and the Princeton Borough Council. He was a veteran of World War I and a past commander of Princeton Post 76 of the American Legion.

Dr. Silvester practiced in Princeton from 1919 to 1942, after which he worked for the State Department of Health for ten years.

★**Oscar Wennergren** (OSU '12), 68, Logan, Utah, a civic and religious leader, died in a Logan hospital after a long illness.

Dr. Wennergren had been meat inspector and state deputy brands inspector in Logan. He was twice president of the Utah V.M.A., president of the Intermountain Livestock Sanitary Association, and an officer of the Logan Canyon Cattlemen's Association. He had also served as president of the Logan Rotary Club and of the Young Men's Mutual Improvement Association.

Surviving Dr. Wennergren are his widow, Mrs. Oscar Izatt Wennergren, two daughters, and two sons.

★**Robert R. Watkins** (TEX '41), 44, Verona, N. J., died Jan. 29, 1958, following a heart attack shortly after he returned from playing golf.

He is survived by his widow, Mrs. Patricia Norton Watkins.

• • •

Other Deaths Reported.—The following deaths have been reported. The usual information for an obituary was not supplied.

Donald J. Campbell (ONT '11), Shawville, Que., died Dec. 12, 1957.

Albert A. Clark, Marianna, Fla., died in April, 1958.

R. R. House, Kinsley, Kan., died Feb. 24, 1958.

★William R. Hulbush (WSC '38), 47, Santa Maria, Calif., died in March, 1958.

Volney C. McCrary, 95, Brevard, N. Car., died May 4, 1958.

★L. E. Mosbach (ISC '30), 50, Humboldt, Iowa, died April 1, 1958.

Carl Olson (CVC '03), Sac City, Iowa, died April 1, 1958.

Merrit Pennington, 63, Dale, Ind., died April 16, 1958.

Frank E. Theobald (CIN '09), 72, Dayton, Ohio, died March 23, 1958.

Boyd B. Tucker, Jr., (TEX '41), 41, Evansville, Ind., died May 23, 1958.

★Lafe J. Waterbury (ONT '50), Menomonee, Wis., died Dec. 20, 1957.

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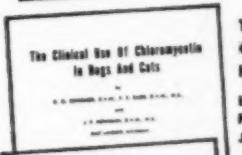

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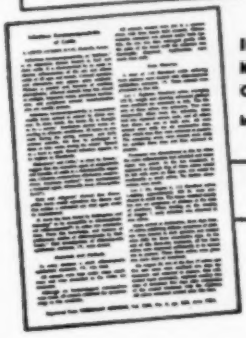

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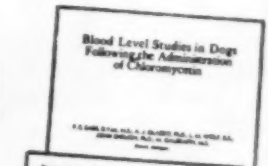
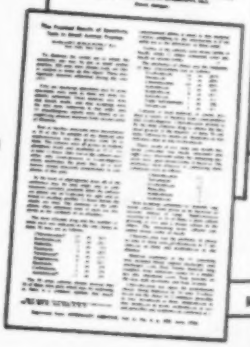




BLOOD LEVEL STUDIES IN DOGS FOLLOWING THE ADMINISTRATION OF CHLOROMYCETIN

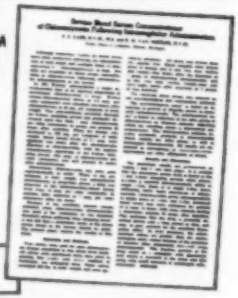
by F. E. Eads, D.V.M., M.S.; A. J. Glazko, Ph.D.; L. M. Wolf, B.S.; John Ehrlich, Ph.D.; M. Galbraith, M.S.

Reprint from the American Journal of Veterinary Research

THE PRACTICAL RESULTS OF SENSITIVITY TESTS IN SMALL ANIMAL PRACTICE

by Margaret Schlichting, B.A.

Reprint from Veterinary Medicine



BOVINE BLOOD SERUM CONCENTRATIONS OF CHLOROMYCETIN FOLLOWING INTRAMUSCULAR ADMINISTRATION

by F. E. Eads, D.V.M., M.S. and K. D. Van Hecker, D.V.M.

Reprint from The California Veterinarian

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Secretary—Mrs. A. W. Eivers, 1595 N. 18th, Salem, Ore.

Recipients of 1958 Achievement Awards Given by the AVMA Women's Auxiliary.—A cash prize and a certificate is presented each year by the Women's Auxiliary to the AVMA to a senior student of each accredited school of veterinary medicine in the United States and Canada.

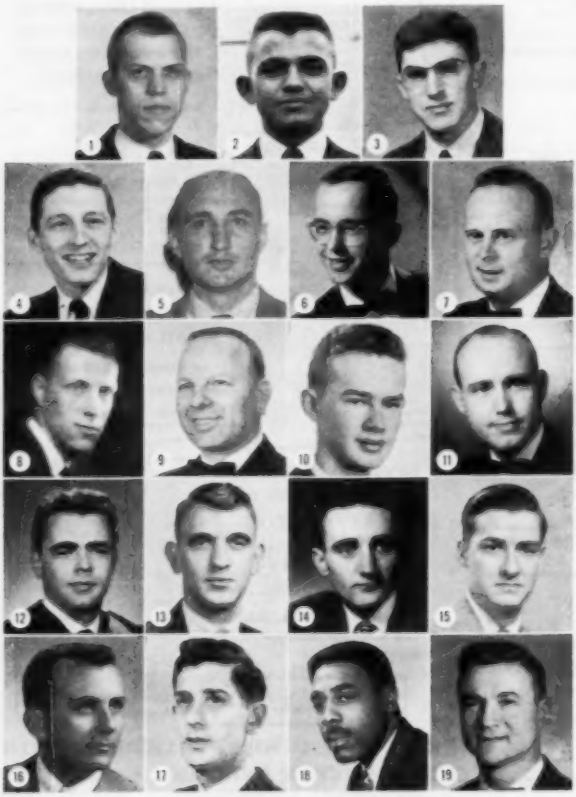
The student is chosen on the basis of activities tending to further the prestige of veterinary medicine. Selection of this student is made by the dean and faculty or by the student chapter of the AVMA.

Many of these young men have accomplished a great deal in the field of research and have done much toward helping to advance veterinary medical research.

Others have done outstanding work and held responsible positions in other phases of campus activities. Their extracurricular activities are many and varied, but all are equipped with a quality of leadership that increases the recognition and the prestige of veterinary medicine.

We congratulate each recipient, and extend to each our sincere wish for a successful career in the profession.

s/(Mrs. J. I.) Elizabeth A. Cornwell,
Second Vice-President.



1) James Carroll Woodard
Alabama Polytechnic Institute
2) Samuel J. Rogers
University of California
3) Kaye E. Wilkins
Colorado State University

4) Donald Wirth
Cornell University
5) Carl Bird
University of Georgia
6) John Edward Schmidt
University of Illinois
7) Warren E. Bohnhoff
Iowa State University

8) James D. Smith
Kansas State College
9) Wayne Wickert
Michigan State University
10) Donald H. Hastings
University of Minnesota
11) Alanson L. Jones
University of Missouri

12) Jacques Webel
University of Montreal
13) Phillip A. Linerode
Ohio State University
14) Robert Godby
Oklahoma State University
15) Richard L. Cook
University of Pennsylvania

16) Charles Farrell Wilson
Texas A. & M. College
17) Michael John Walcroft
University of Toronto
18) Dean R. Hodges
Tuskegee Institute
19) Donald H. Jenkins
Washington State College

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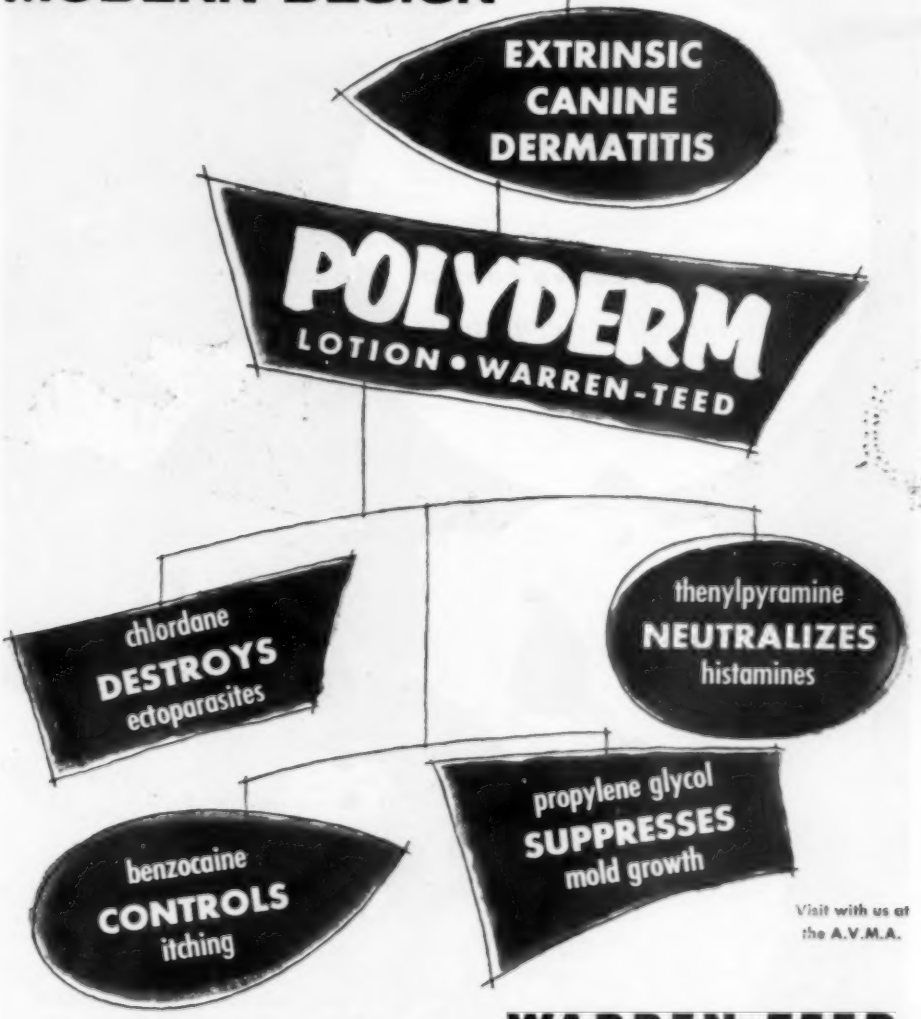

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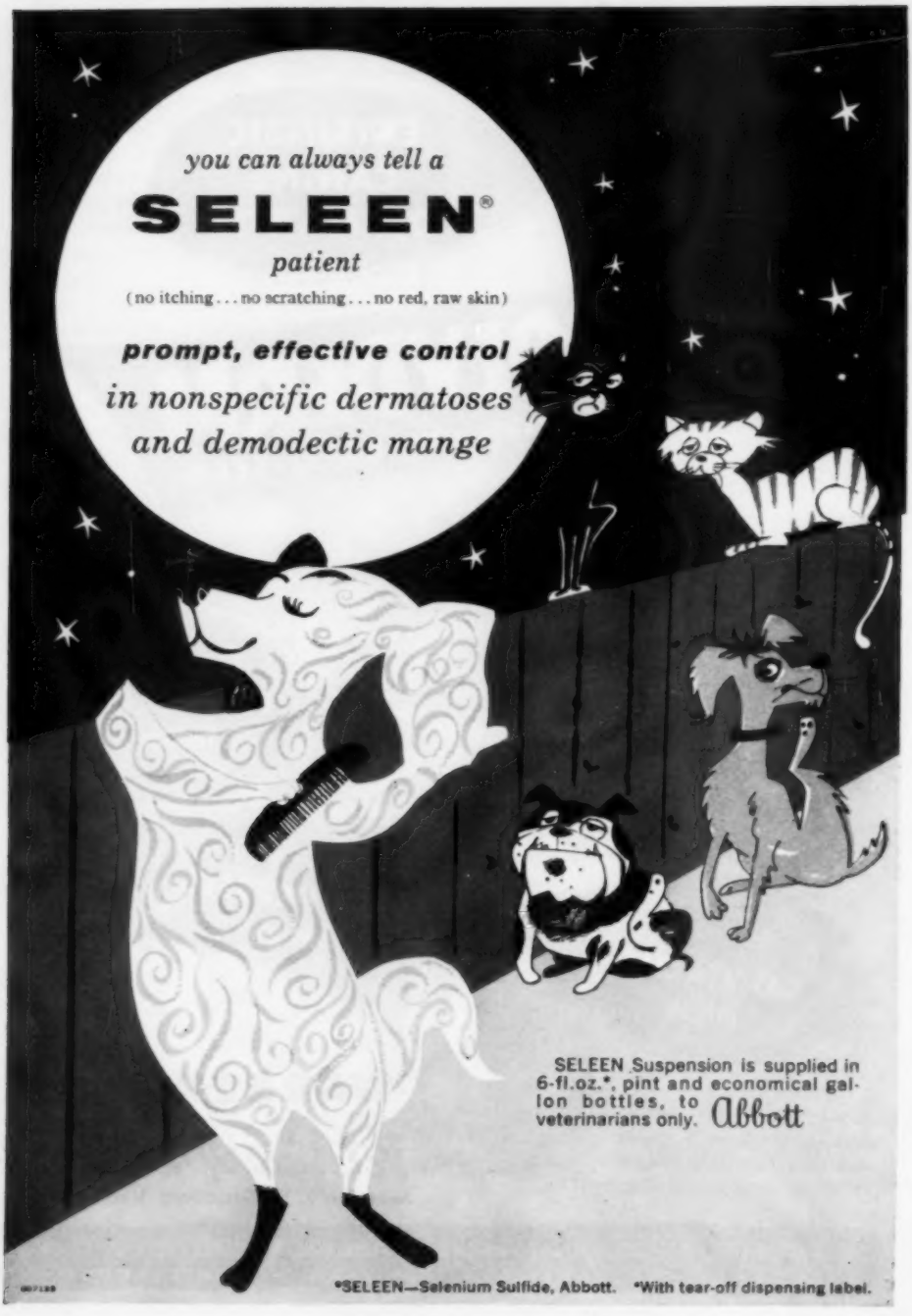
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COMING MEETINGS

Nebraska State Veterinary Medical Association. Summer meeting. Kearney, Neb., July 18-20, 1958. W. T. Spencer, 1250 N. 37th, Lincoln, secretary.

Alabama Polytechnic Institute. Annual conference for veterinarians. School of Veterinary Medicine, Auburn, July 20-23, 1958. T. C. Fitzgerald, program chairman.

Kentucky Veterinary Medical Association. Forty-seventh annual convention. Seelbach Hotel, Louisville, July 21-22, 1958. R. H. Singer, 136 Shawnee Place, Lexington, secretary-treasurer.

Canadian Veterinary Medical Association. Tenth annual Convention. Royal Alexandra Hotel, Winnipeg, Manitoba. July 21-25, 1958. Claude Kealey, 1195 Wellington St., Ottawa 3, Ont., executive secretary.

Iowa State College. Annual conference for veterinarians. Memorial Union, Iowa State College, Ames, July 22-23, 1958. J. B. Herrick, extension veterinarian.

Connecticut Veterinary Medical Association. Annual outing meeting. Rosewood Inn and Grove, Simsbury, Conn., Aug. 6, 1958. E. H. Patchen, 289 New Haven Ave., Milford, Conn., secretary.

College of Veterinary Surgeons of Province of Quebec. Annual meeting. Quebec, Aug. 8-9, 1958. Jacques Saint Georges, St. Hyacinth, Que., public relations committee.

Mississippi State Veterinary Medical Association. Fifty-second annual meeting. Buena Vista Hotel, Biloxi, Aug. 10-12, 1958. H. F. McCrory, State College, Miss., secretary.

American Association of Veterinary Bacteriologists. Annual meeting. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Aug. 16, 1958. C. H. Cunningham, Michigan State University, College of Veterinary Medicine, East Lansing, secretary.

American Veterinary Medical Association. Ninety-Fifth Annual Meeting. Convention Hall, Philadelphia, Pa., Aug. 18-21, 1958. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

Louisiana Veterinary Medical Association, Inc. Annual meeting. Montelone Hotel, New Orleans, Aug. 24-26, 1958. R. R. Gibbs, 6421 Chef Menteur Highway, New Orleans, La., program chairman.

New York State Veterinary Medical Society. Sixty-seventh annual meeting. Concord Hotel, Kiamesha Lake, N. Y., Sept. 4-6, 1958. Miss Joan S. Halat, 803 Varick St., Utica, N. Y., executive secretary.

Washington State Veterinary Medical Association. Annual meeting. Hotel Olympian, Olympia, Sept. 8-10, 1958. F. M. Shigley, P.O. Box 1163, Olympia, Wash., program chairman.

South Dakota Veterinary Association. Meeting. Hotel Cateract, Sioux Falls, Sept. 16-17, 1958. G. E. Duncan, Tyndall, secretary.

New England Veterinary Medical Association. Annual meeting. Hotel Wentworth-By-The-Sea, Portsmouth, N. H., Sept. 28-Oct. 1, 1958. C. Lawrence Blakely, 180 Longwood Ave., Boston 15, Mass., secretary-treasurer.

Missouri, University of. Annual short course for graduate veterinarians. School of Veterinary Medicine, Columbia, Mo., Oct. 6-7, 1958. Cecil Elder, chairman.

Florida State Veterinary Medical Association. Annual meeting. Galt Ocean Mile Hotel, Fort Lauderdale, Oct. 12-14, 1958. A. R. Chambers, 6116 Main St., Jacksonville, secretary.

District of Columbia Veterinary Medical Association. Annual all-day meeting. Walter Reed Army Medical Center, Washington, D.C., Oct. 14, 1958. W. I. Gay, 5200 Chandler St., Bethesda, Md., secretary-treasurer.

Eastern Iowa Veterinary Association, Inc. Annual meeting. Hotel Roosevelt, Cedar Rapids, Oct. 16-17, 1958. F. E. Brutsman, Traer, Iowa, secretary-treasurer.

Texas Veterinary Medical Association. Annual meeting. Stephen F. Austin Hotel, Austin, Oct. 19-21, 1958. Paul B. Blunt, 710 Maverick Bldg., San Antonio, secretary.

Southern Veterinary Medical Association. Annual meeting. Claridge Hotel, Memphis, Tenn., Oct. 26-30, 1958. A. A. Husman, P.O. Box 91, Raleigh, N. Car., secretary.

Arizona Veterinary Medical Association. Annual meeting. Yuma, Ariz., Dec. 7-9, 1958. R. E. McComb, Jr., 4730 N. 7th Ave., Phoenix, program chairman.

Tennessee Veterinary Medical Association. Annual meeting. Noel Hotel, Nashville, Jan. 11-13, 1959. H. W. Hayes, 5009 Clinton Pike, Knoxville, secretary-treasurer.

Oklahoma Veterinary Medical Association. Annual meeting. Mayo Hotel, Tulsa, Jan. 25-27, 1959. M. N. Riemenschneider, 122 State Capitol Bldg., Oklahoma City, secretary.

Ohio State Veterinary Medical Association. Annual convention. Neil House Hotel, Columbus, Feb. 4-6, 1959. Harry C. Sharp, 1411 W. Third Ave., Columbus, Ohio, executive secretary.

Foreign Meetings

Sixth International Congresses on Tropical Medicine and Malaria. Lisbon, Portugal, Sept. 5-13, 1958. Professor Manuel R. Pinto, Institute of Tropical Medicine, Lisbon, secretary-general. (Membership application forms may be obtained by U.S. veterinarians by writing to the AVMA.)

German Veterinary Association. Regular biennial meeting. Hannover, Germany, Sept. 19-21, 1958. Dr. Karl Ohly, 123 Forsthausstrasse, Frankfurt/Main, Germany, president.

Seventh International Congress on Hydatid Disease, Beirut, Lebanon, Sept. 20-22, 1958. Dr. Elias Sader, c/o Lebanese Order of Physicians, P.O.B. 640, Beirut, Lebanon, secretary-general. (Applications to present papers must be accompanied by an abstract).

International Veterinary Congress. Sixteenth session. Madrid, Spain, May 21-27, 1959. Prof. Pedro Carda A., general secretary. Calle Villanueva 11, Madrid.

U.S. COMMITTEE: Dr. W. A. Hagan, chairman, New York State Veterinary College, Ithaca, N. Y.; Dr. J. G. Hardenbergh, secretary, 600 S. Michigan Ave., Chicago 5, Ill.

Third World Congress on Fertility and Sterility. Amsterdam, Holland, June 7-13, 1959. Dr. L. I. Swaab, Sint Agnietenstraat 4, Amsterdam, Holland, honorary secretary.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. Dr. G. W. Jones, Main St., Prattville, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

North Alabama Veterinary Medical Association, the sec-

ond Thursday of November, January, March, May, July, and September, in Decatur, Ala. Ray A. Ashwander, Decatur, Ala., secretary.

North East Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2 Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldson, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16 St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. James L. Frederickson, 17 Niles St., Bakersfield, Calif., secretary-treasurer.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643-N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. M. Granfield, 2600 W. El Camino Real, San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert L. Chandler, P.O. Box 8, Ukiah, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Chapter SCVMA, the second Tuesday of each month at 7:30 p.m., Hody's Restaurant, North Hollywood, Calif. Dr. V. H. Austin, 14931 Oxnard St., Van Nuys, secretary-treasurer.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. Dr. Rolf Reese, 23815 Ventura Blvd., Calabasas, Calif., secretary.

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Dr. E. C. Jones Honored by Kansas State College

Dr. Eddell Charles Jones, president of Norden Laboratories, Lincoln, Neb., was given a distinguished Service Award in veterinary medicine by his alma mater, Kansas State College, on June 6, 1958. The college presented the award at a banquet held during the twentieth annual conference for Kansas veterinarians in Manhattan.

Dr. Jones, a 1916 graduate, has been president and chairman of the board of directors of Norden

Dr. E. C. Jones

Laboratories since 1954. For more than three decades Dr. Jones made major contributions in developing quality serums, vaccines, and drugs for the veterinary medical profession. He helped to organize the Platte Valley Serum Company, Grand Island, Neb., later serving as president of the firm. This company merged with Norden Laboratories in 1934, at which time Dr. Jones became vice-president and sales manager.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinary Medical Association, the second Thursday of each month. D. E. Britten, 544 N. Ben Maddox, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyer, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. William F. Casler, 2540 30th Ave., N., St. Petersburg, secretary-treasurer.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4140 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla. secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Donald C. Ford, Forest Park, secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Charles H. Armstrong, 1021 Davis St., Evanston, secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. Alfred G. Schiller, Veterinary Clinic, University of Illinois, Urbana, secretary-treasurer.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at

the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P.O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Association, the second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. Dr. J. G. Irwin, Iowa City, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each month, September through May, at 6:30 p.m., at the Gardson Hotel, Estherville, Iowa. Barry Barnes, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort Dodge, Iowa. H. Engelbrecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer, Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October, Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, Creston, secretary.

Upper Iowa Veterinary Medical Association, the third Tuesday of each month at 7:00 p.m., at All Vets Center, Clear Lake, Iowa. Richard Baum, Osage, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Varsailles Rd., Frankford, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Norman Herbert, 3506 Joann Drive, Baltimore 7, Md., secretary.

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REFERENCES: 1. Bull, W. S.: N. Amer. Vet., in press. 2. Henry, R. T., and Blackburn, E. G.: Vet. Med., in press.

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Make your diagnosis from the picture below—then turn the page ▶

Figure 1

History.—A male Airedale, 10 years old, had been "dribbling" urine for about a week. Polydipsia was also reported and a urinous odor from the breath was noticeable. A large mass could be palpated in the posterior part of the abdomen. A pneumocystograph was made.

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—A smooth mass in the area of the prostate, which has caused the urinary bladder to be pushed anteriorly.

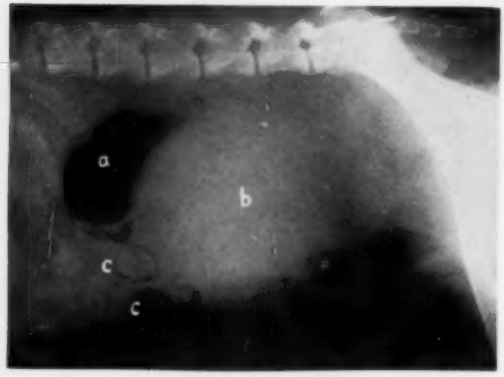

Fig. 2—Radiograph of lateral view of abdomen, showing (a) inflated urinary bladder; (b) cystic prostate; and (c) segments of small intestines.

Comment (Dr. G. B. Schnelle, Boston, Mass.)—The air injected via the catheter depicts a small, compressed bladder (fig. 2) anterior to the large, smooth mass which extends from the pelvis forward. At the time of operation this proved to be a cyst originating in the prostate. The exact nature of these cysts is not well understood. They can be of embryonal origin, neoplastic, or due to cystic degeneration of a large prostate into which urine seems to escape, causing fairly rapid enlargement of the organ. These lesions are rare and whether they are all of the same nature or origin is not known. Surgical excision is possible, and has been done successfully.

This case was submitted by the staff of the Angell Memorial Animal Hospital, Boston, Mass.

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MICHIGAN—Central Michigan Veterinary Medical Association, the first Wednesday of every month at 7 p.m. Frank A. Carter, P.O. Box 78, Carson City, Mich., secretary.

Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Edwin E. Epstein, 4877 Natural Bridge Ave., St. Louis 15, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Frank A. O'Donnell, Parksville, Mo., secretary-treasurer.

NEVADA—Western Nevada Veterinary Society, the first Tuesday of each month. Paul S. Silva, 1170 Airport Road, Reno, Nev., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. Burritt Lupton, 569 Franklin Ave., Wyckoff, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. R. M. Sauer, secretary.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, third Wednesday of each month, Fez Club, Albuquerque, N.M. Jack Ambrose, 3018 N. Rio Grande Blvd., Albuquerque, secretary-treasurer.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College, Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified

monthly, Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. T. L. James, Box 243, Newton, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leak St., Southern Pines, N. Car., secretary.

Western North Carolina Veterinary Medical Association, the second Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Ashville, N. Car. Vilu Lind, 346 State St., Marion, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuller's Wigwam, 6210 Hamilton Ave., at North Bend Road. G. C. Lewis, Cincinnati, Ohio, secretary-treasurer.

Columbus Academy of Veterinary Medicine, every month, September through May. E. M. Simonson, Columbus, Ohio, secretary-treasurer.

Cuyahoga County Veterinary Medical Association, the first Wednesday in September, October, December, February, March, April and May, at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. F. A. Coy, Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month. O. W. Fallang, Dayton, secretary.

Killbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginning with February. D. J. Kern, Killbuck, Ohio, secretary-treasurer.

Mahoning County Veterinary Medical Association, the third Tuesday of each month, at 9:00 p.m., Youngstown Maennerchor Club, Youngstown, Ohio. Sam Segall, 2935 Glenwood Ave., Youngstown, secretary.

Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Veterinary Medical Association, the last Wednesday of March and July. C. S. Alvanos, Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge, Canton, Ohio. M. L. Willen, 4425 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m., Ireland's Restaurant, Lloyds', 718 N.E. 12th Ave., Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Willamette Veterinary Medical Association, the third Tuesday of each month, except July and August, at the

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Marion Hotel, Salem. Marvin M. Corff, McMinnville, Ore., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania, School of Veterinary Medicine. Raymond C. Snyder, N.E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blackburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9537 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. I. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Pritchard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). R. J. O'Hern, P.O. Box 617, Cumberland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

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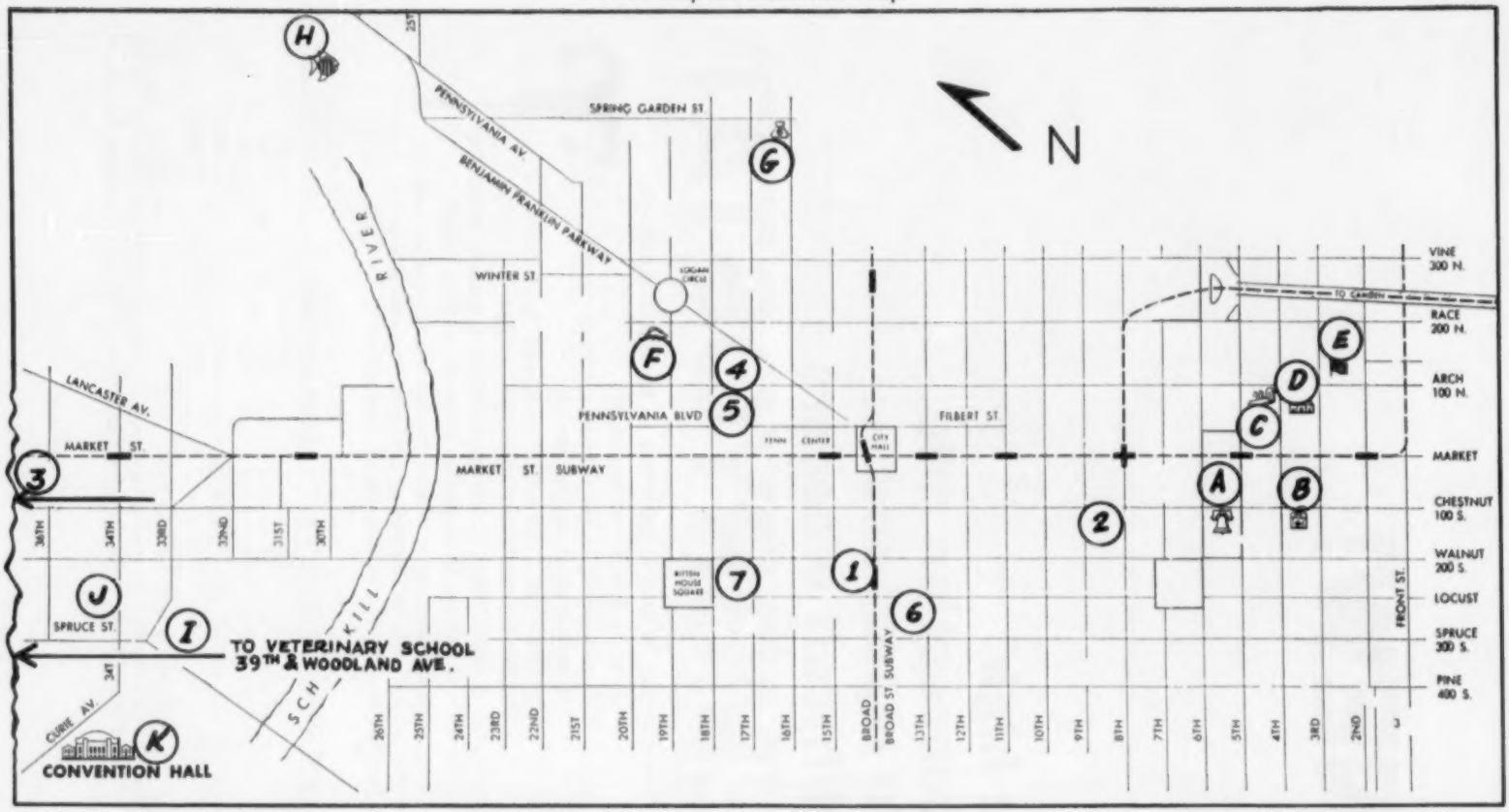

1. Bellevue-Stratford Hotel
2. Benjamin Franklin Hotel
3. Penn-Sherwood
4. Robert Morris (not pictured)
5. Sheraton Hotel
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A. Independence Hall
B. Carpenters' Hall
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F. Academy of Natural Sciences
G. U.S. Mint (Guided Tours)
H. Aquarium
I. University Museum
J. University of Pennsylvania
K. Convention Hall

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Ninety-Fifth Annual AVMA Meeting, Aug. 18-21, 1958

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6. Sylvania ††	7.50- 9.50	.....	10.00-13.00	25.00-27.50*
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Desire position with firm who needs veterinarian experienced in general practice, clinical chemistry, x-ray, and electronics. Address "Box L 34," c/o JOURNAL of the AVMA.

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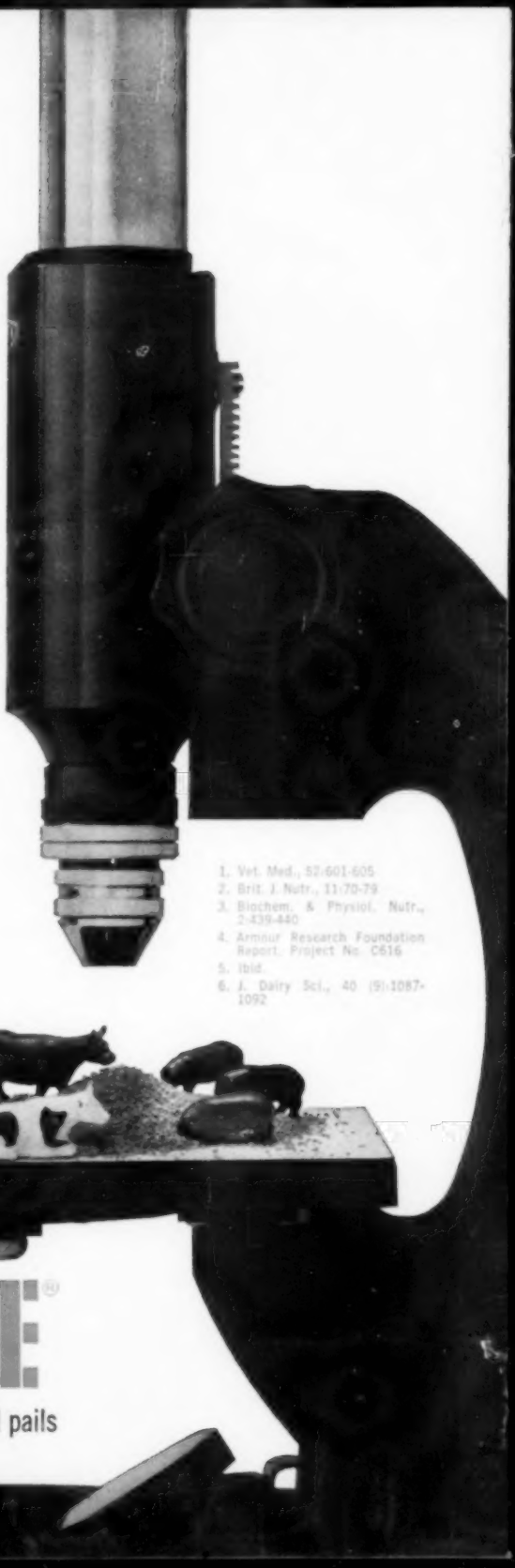

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2. Brit. J. Nutr., 11:70-79
3. Biochem. & Physiol. Nutr., 2:439-440
4. Armour Research Foundation Report, Project No. C616
5. Ibid.
6. J. Dairy Sci., 40 (9):1087-1092

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